The American Journal of Surgery

EDITED BY THURSTON SCOTT WELTON, M.D.

February 1943

TRAUMATIC SURGERY

Papers Read at the June 1942
Meeting of The American Association
for the Surgery of Trauma

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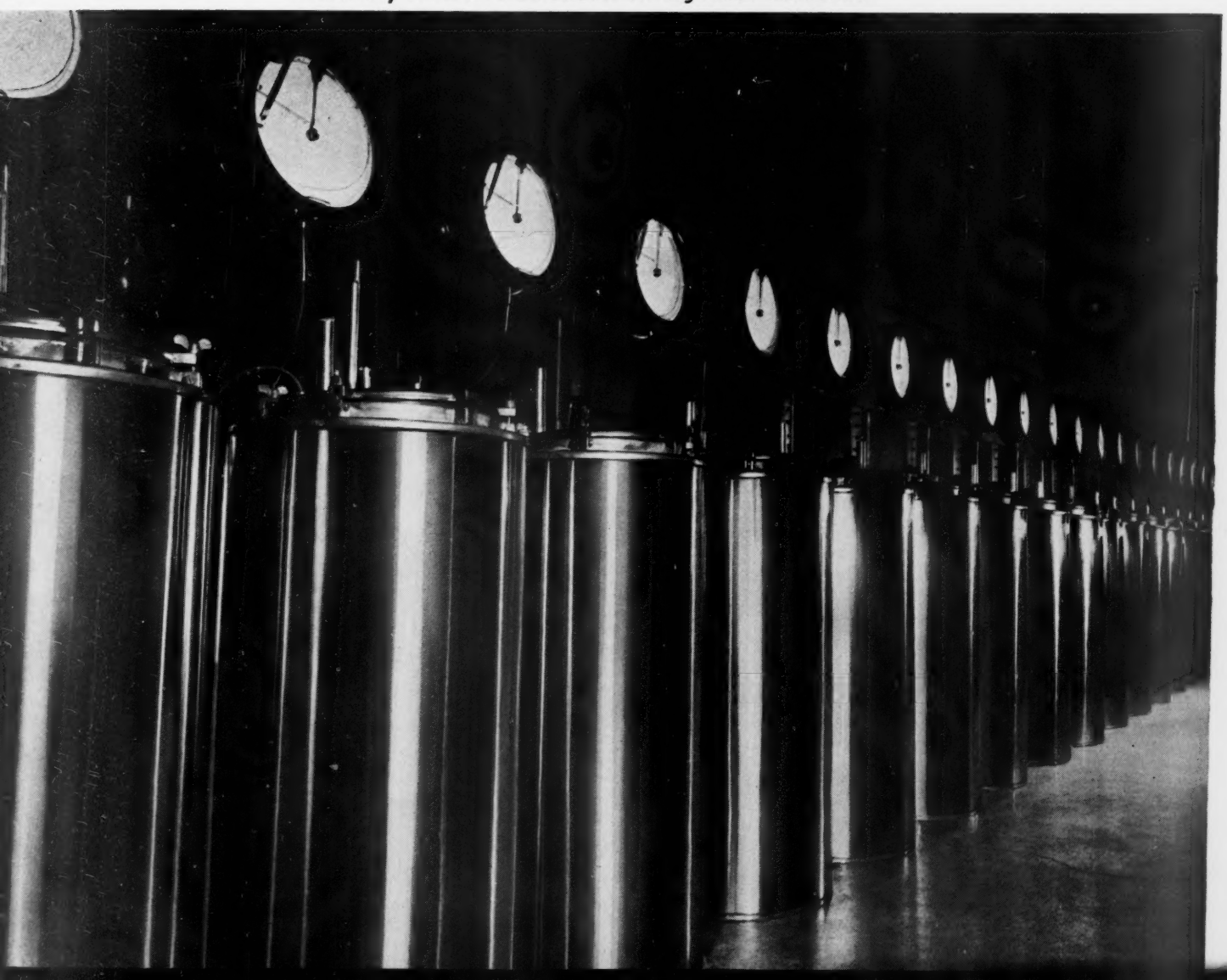

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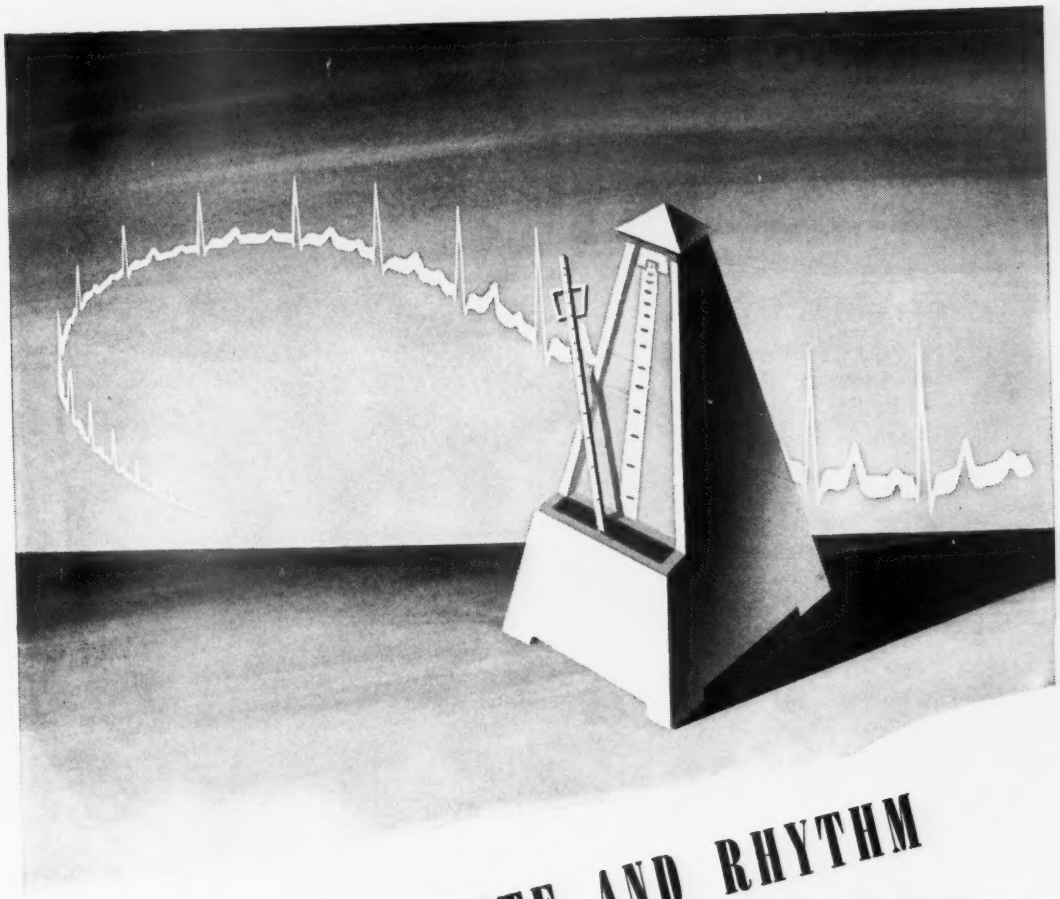

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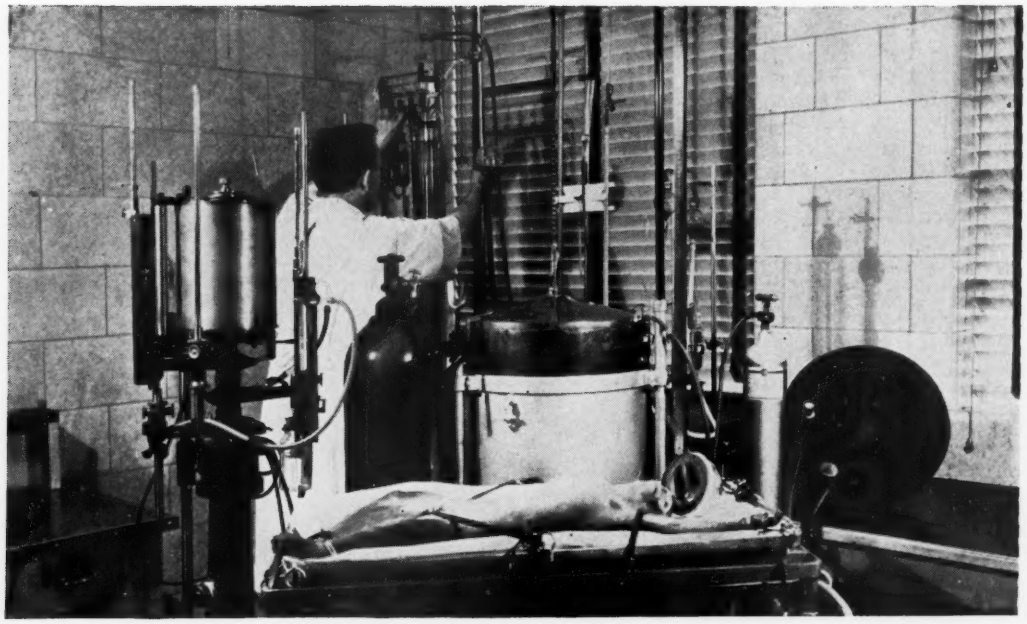

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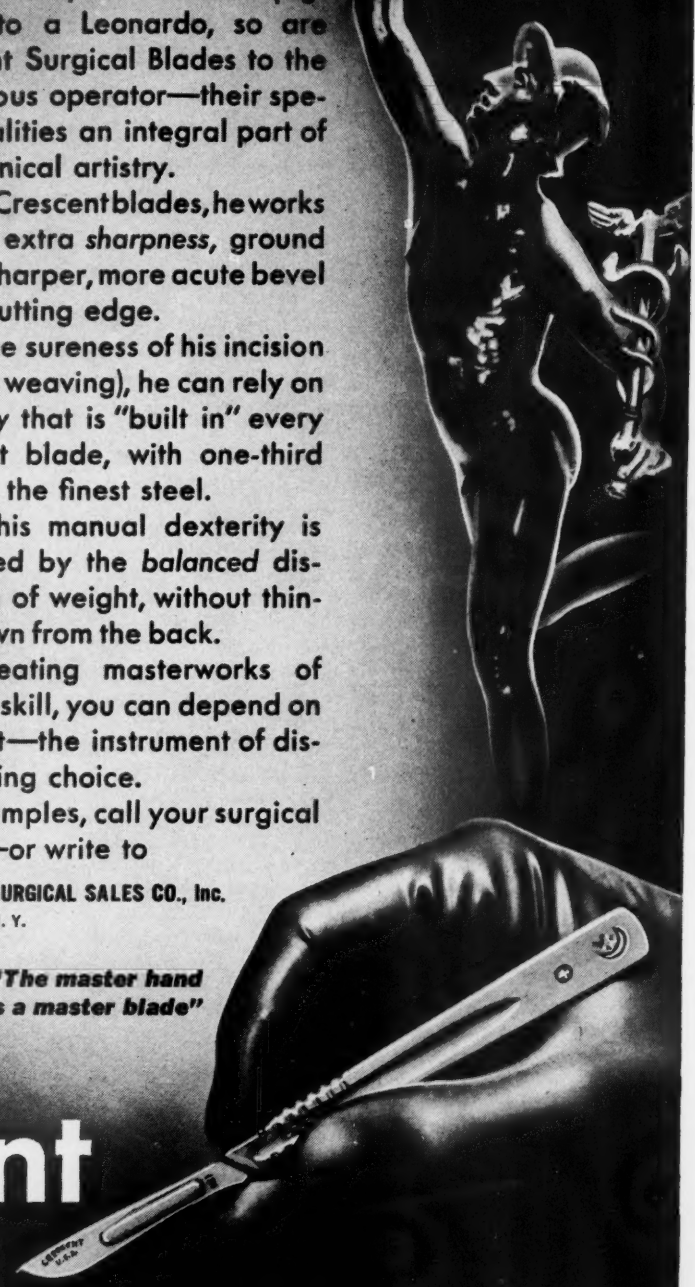

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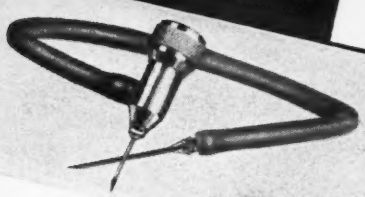

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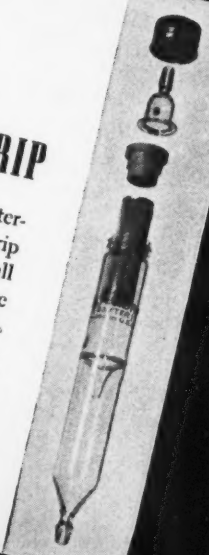

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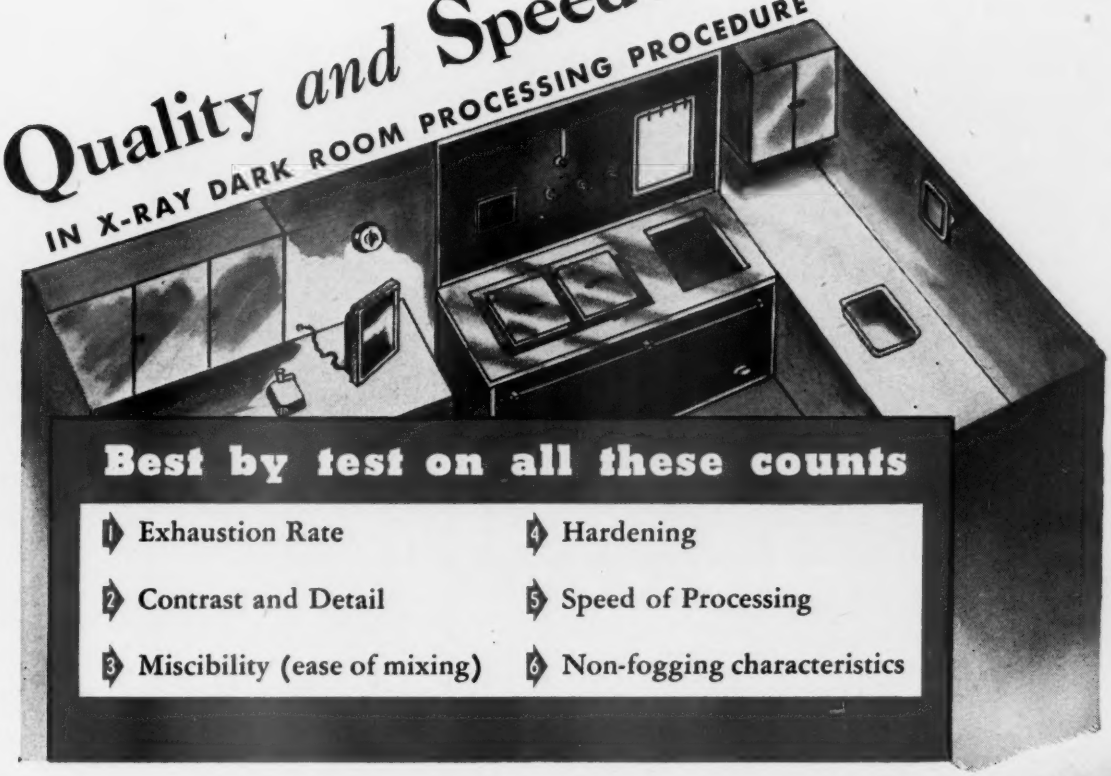

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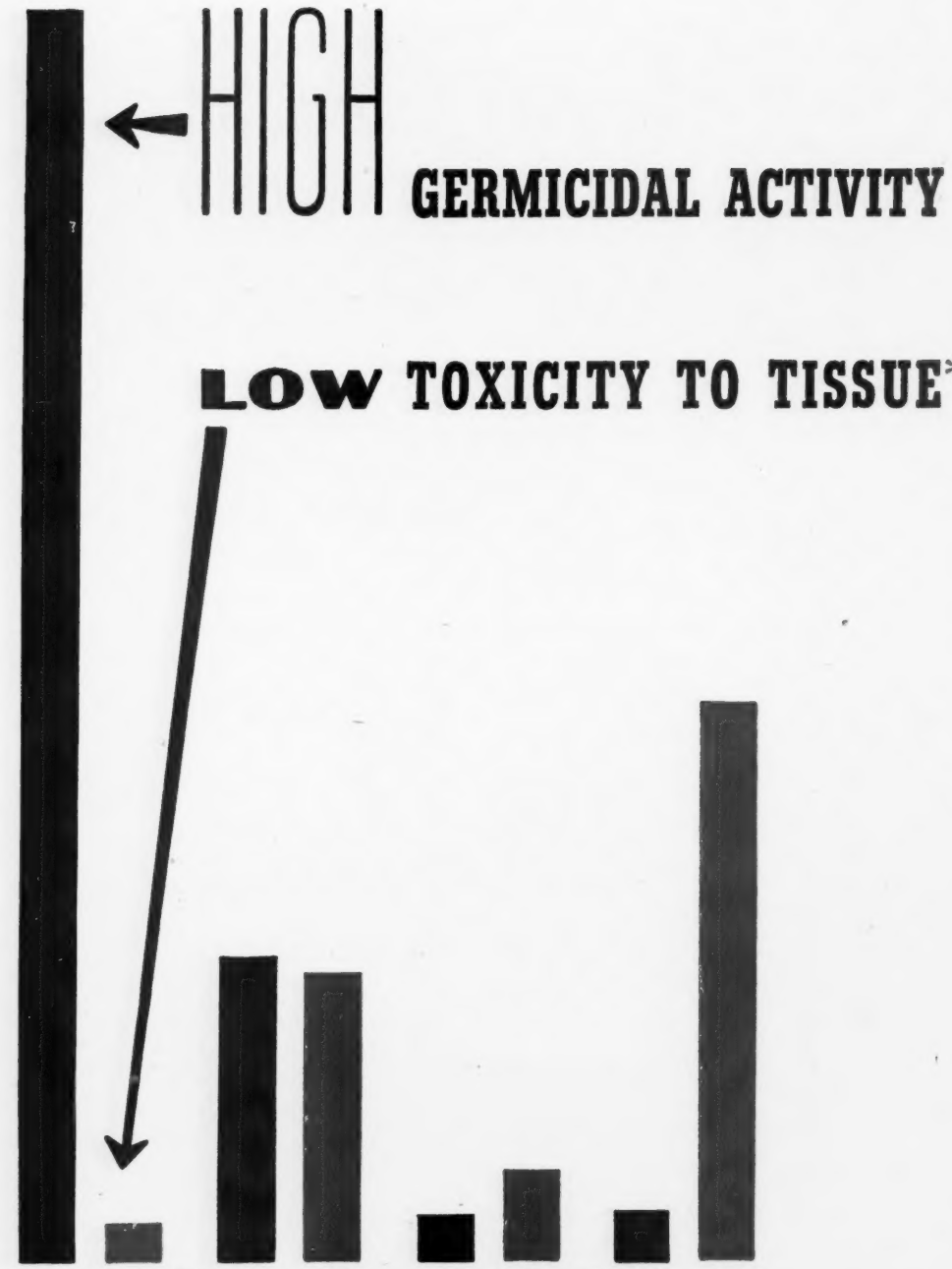

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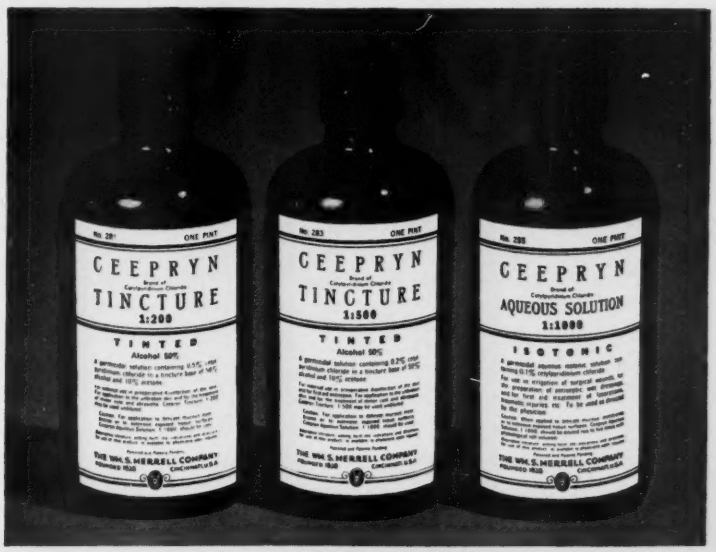

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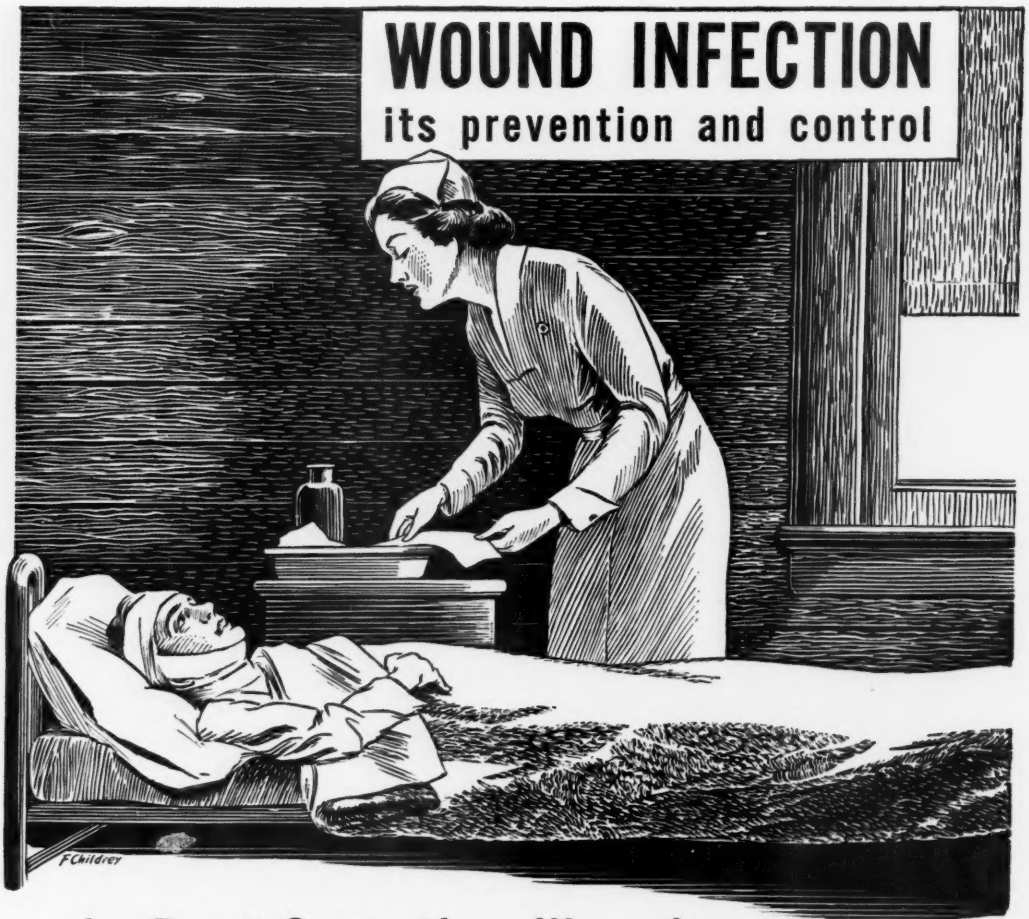

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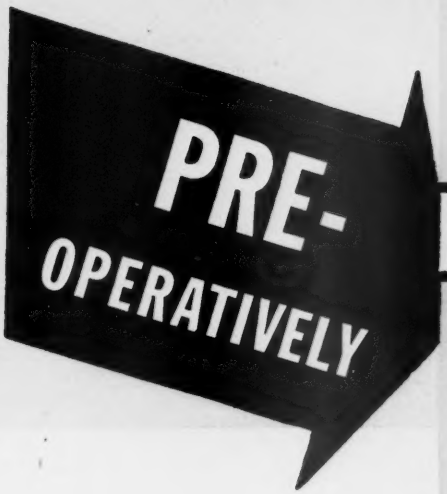

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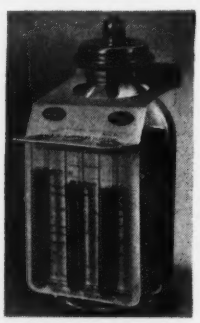

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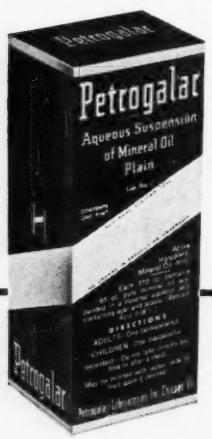

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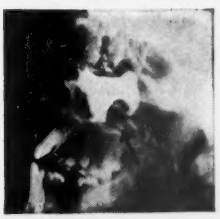

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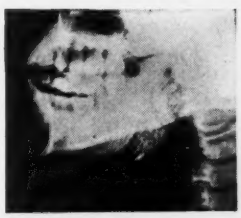

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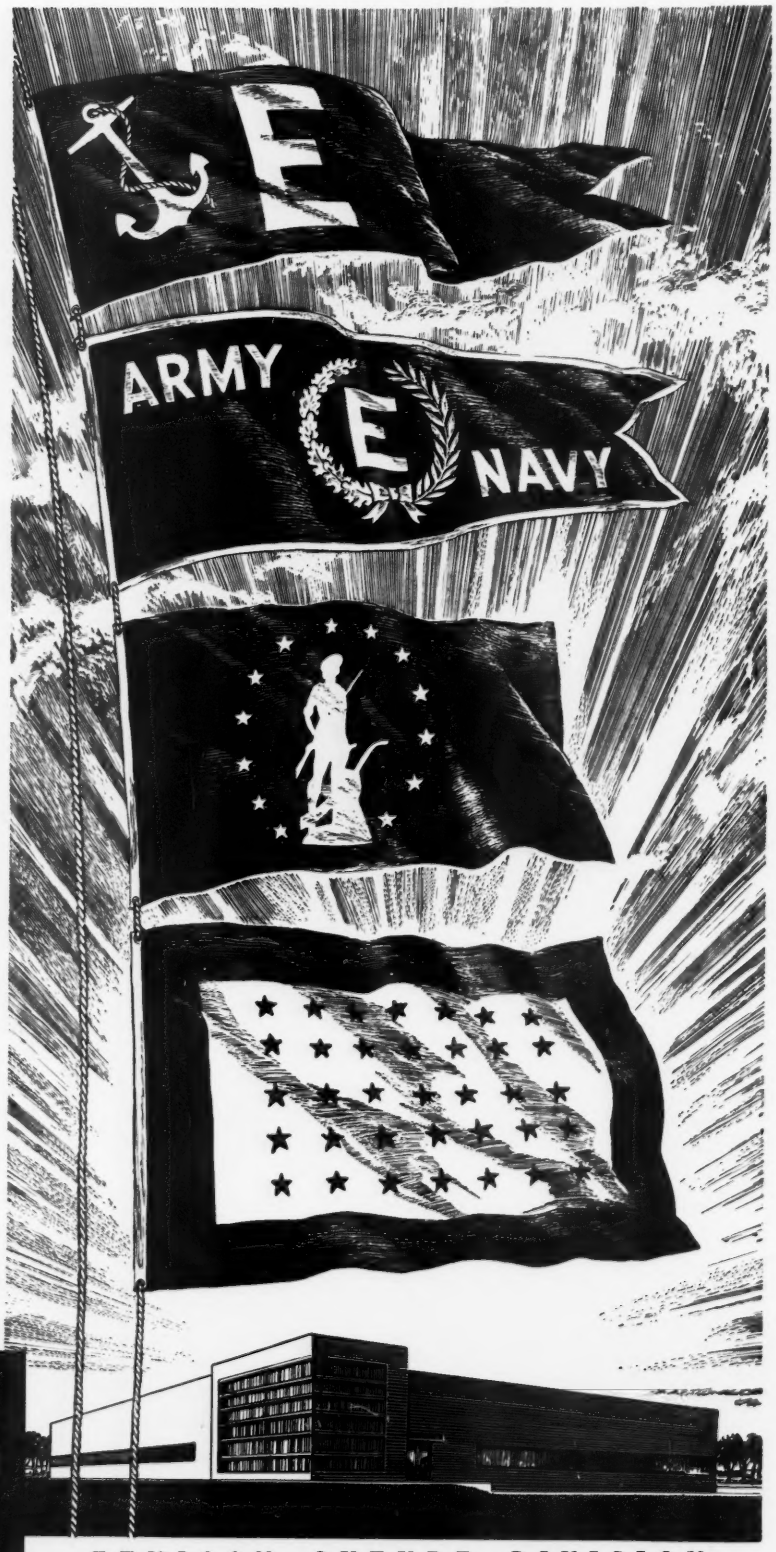

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NEW SERIES VOL. LIX FEBRUARY, 1943 NUMBER TWO

Editorial

OUR SURGEONS IN THE PRESENT WAR

PRESIDENTIAL ADDRESS

HENRY C. MARBLE, M.D.

BOSTON, MASSACHUSETTS

TWENTY-FIVE years ago, when the United States entered the first World War, the surgeons of this country were called upon to wrest themselves from their peace-time practices and join with their countrymen on the surgical staffs of our fighting forces. At that time we had had very little training for the task that was before us. Although the surgery of wounds is the oldest branch of medicine, the surgeons of our era had been so much concerned with abdominal, gynecological and cranial technics that little time had been given to the teaching of this ancient art. It may have been that the optimism of peace and the inability of most of us to imagine a world-wide war in a time of apparent enlightenment was responsible for this neglect.

When we were confronted with the emergency, we hurriedly adopted from the French and the English, who had been deep in this work for over two years, all that we believed to be good and we applied this borrowed knowledge in the care of our own wounded men. What we lacked in training and experience we tried to overcome by willingness and application of sound fundamentals. The end results of our efforts and the consequences of our failures are medical history.

After the Armistice we returned to our peace-time practices but not to our previous neglect of the study and teaching of the surgery of injuries. Realization came to the war surgeon that the problems of

war and peace are not foreign to one another; that the difference is only a change in location; the people and problems are the same.

During the past twenty years industry has expanded, the motor car has become popular, speed has increased and accidents are more numerous. Our hospital clinics are filled with the injured and wards are set aside for their care. The war surgeon continuing his interest in the surgery of trauma, offered his knowledge and experience to the younger staff members, and through research with them developed new medicines, means and methods.

And now another World War is upon us and thousands of young surgeons have again been called upon to care for the war wounded. Surely the surgeon going out today is far better trained, has a deeper knowledge and more mature judgment than his older brother of a quarter century ago. The soldier going into action today knows that he will be cared for by the finest trained and equipped corps of doctors that his country can give him and that, if he is wounded, his chances of recovery are greater than ever before. Knowing this he will do his job better.

The latest step in this educational trend is the organization of the American Association for the Surgery of Trauma, and its duty is clear. We must maintain a thinking membership to administer and direct; we must so plan our councils that we may receive, evaluate and judge all that is new in wound surgery. We must welcome all who are interested and qualified to the end that this knowledge may be spread to the profit of all. In war even more than in peace we have our work to do.

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Original Articles

SUPRACONDYLAR FRACTURES IN CHILDREN

ALEXANDER P. AITKEN, M.D.,
Junior Visiting Surgeon, Boston City Hospital
BOSTON, MASSACHUSETTS

LYMAN SMITH, M.D.
Formerly Resident Surgeon, Boston City Hospital
BARRINGTON, ILLINOIS

AND

CHARLES W. BLACKETT, M.D.
BOSTON, MASSACHUSETTS

ONE of the most difficult fractures to treat in childhood is the supracondylar fracture of the humerus. These fractures are often not only difficult to reduce, but are difficult to maintain in position. It is the general belief that accurate reduction in children is not essential for a good result. It is true that the functional end results of malalignment are generally very good, but it is also true that the cosmetic end results are often very poor. No patient can be expected to be satisfied with an excellent functional result if he also has a cubitus varus deformity. Our experience at the Boston City Hospital with the accepted forms of treatment has not been satisfactory. Recurrence of displacement has occurred despite accurate reduction and immobilization in flexion either by means of adhesive strapping or plaster splints. This has been particularly prone to occur in cases showing ulnar displacement with rotation of the distal fragment. Because of our dissatisfaction with our methods of treatment, we have made an end result study of fifty cases to determine the results of malalignment and to develop a more satisfactory method of treatment.

In this series of cases, the length of time elapsed between the dates of injury and examination, has not been less than three years and not more than seven years.

The original *x*-rays could not be obtained in one group of twelve cases. The end results of this group are as follows: Six cases are per-

fectly normal, functionally, cosmetically and by *x*-ray. Two cases show slight loss of the carrying angle, one a slight increase of the carrying angle and three a definite cubitus varus deformity.

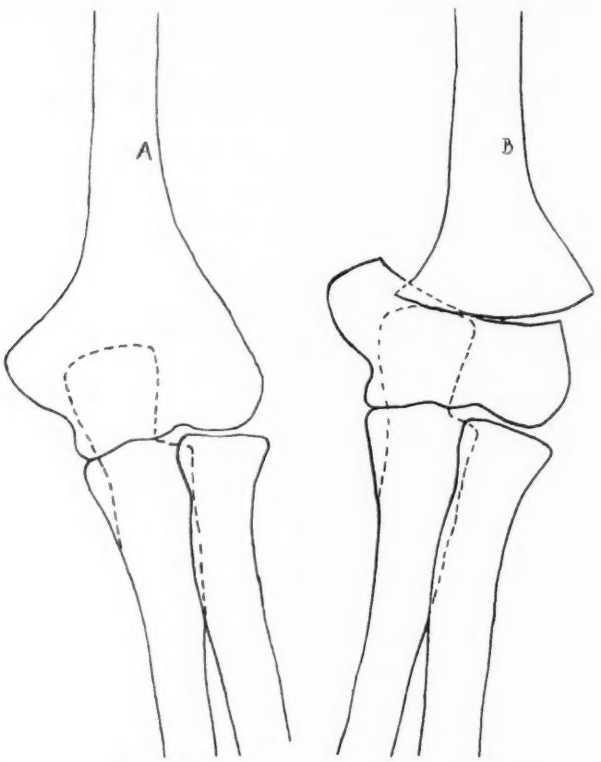


FIG. 1. A, showing normal carrying angle of left elbow. B, showing loss of carrying angle by ulnar displacement of distal fragment.

The original *x*-rays in the remaining thirty-eight cases showed the following types of displacement: No displacement, five cases; posterior and ulnar displacement with rotation of the distal fragment, sixteen cases; posterior and radial displacement with rotation of the distal fragment, eleven cases; posterior displacement with rotation of the distal fragment but without lateral or medial displacement, four cases; anterior displacement, two cases.

The end results of the various types of displacement are as follows.

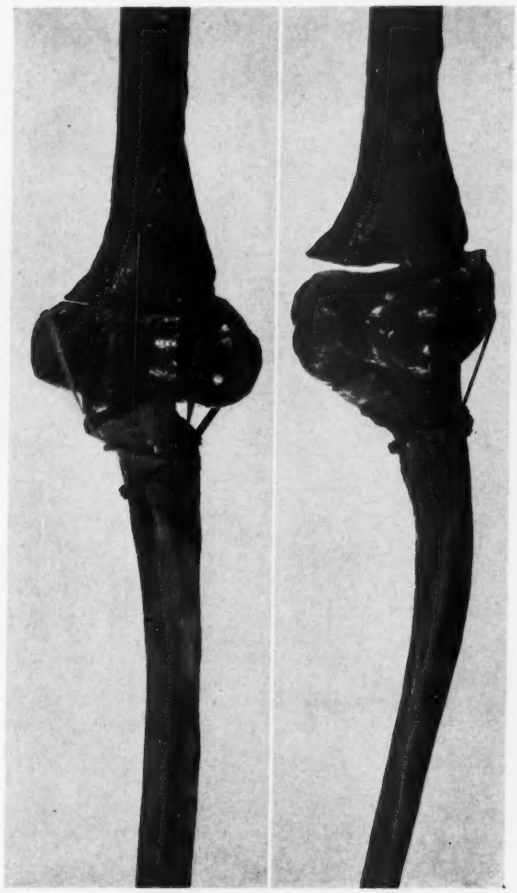
A. Cases originally showing no displacement (five cases).

The original position was maintained in all cases. All now show no evidence of injury functionally, cosmetically or by *x*-ray.

B. Ulnar and posterior displacement with rotation (sixteen cases).

Excellent reduction was obtained and maintained in four cases. All now show excellent results. In nine cases, the displacement either

recurred or was incompletely reduced. All cases united with persistent ulnar and posterior displacement with rotation. All cases developed definite varus deformity. In two cases, the ulnar and



A B

FIG. 2. A, showing normal carrying angle. B, shows loss of carrying angle due to rotation of distal fragment.

posterior displacements were corrected, but rotation of the distal fragment persisted. Both cases now show slight loss of the carrying angle.

One case had none of the components of displacement corrected, yet now shows an excellent result. Although *x*-rays failed to reveal involvement either of the trochlear or capitellar epiphysis by the fracture line, yet the trochlear epiphysis was apparently stimulated. The resulting overgrowth of the medial half of the shaft prevented the development of a varus deformity and brought about a normal

carrying angle. We have shown in previous publications that stimulation of an epiphysis by a remote fracture does occur. In this particular case we feel sure that the normal result was due to

FIG. 3. A, case showing marked ulnar displacement with callus formation. B, same case, one year later, showing considerable varus deformity due entirely to the ulnar displacement.

stimulation of the trochlear epiphysis and not to retardation of the capitellar epiphysis.

C. Radial and posterior displacement with rotation (eleven cases).

An excellent reduction was obtained in only one case, which now shows an excellent result. Only partial reduction of all components was obtained in seven cases. All now show excellent results. In one case posterior displacement and rotation were corrected but radial displacement persisted. An increase in the carrying angle was the end result. Another case had only slight radial displacement but rather marked rotation and now shows a varus deformity of 5 de-

grees. In one case the displacement was corrected from radial displacement to one of ulnar displacement and now shows a marked varus deformity.

D. Posterior displacement and rotation only (four cases).

Two had excellent reductions and now show no evidence of their injury. One had correction of the posterior displacement but not of the rotation and now shows some loss of the carrying angle.

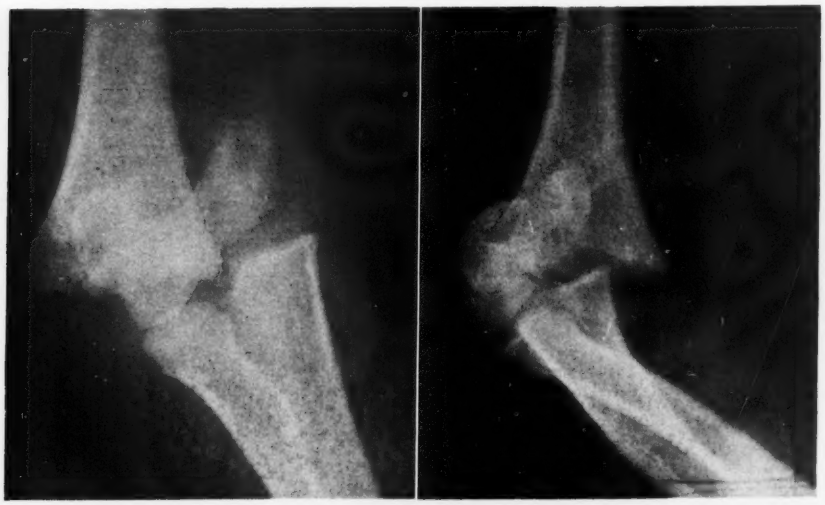
One had slight persistent posterior displacement with considerable rotation and now shows complete loss of the carrying angle.

E. Anterior displacement (two cases).

One case showing radial displacement was not completely reduced but shows now an excellent result. One case showing ulnar displacement was not completely reduced and developed a varus deformity. This case also showed a vertical T-fracture which extended into the capitellar epiphysis. Contrary to what might have been expected, this epiphysis did not show retardation of growth but was stimulated by the fracture as the deformity has increased over a six-year period.

In an excellent article on the end results of supracondylar fracture, Siris presented several cases in which either the trochlear or capitellar epiphyses were involved in the fracture. In his cases the involved epiphyses showed retardation of growth with resulting deformity. Our experience with fractures of the epiphyses has been similar to that of Siris. Stimulation of the epiphysis by the fracture, as in the above cases, is very unusual. Brewster and Karp believe that the varus deformity in such fractures is due to stimulation of the capitellar epiphysis. Such stimulation probably does occur in some cases. Deformity, then, can result either from stimulation or retardation of the epiphyses whether the epiphyses are directly involved in the fracture line or not. However, in the great majority of cases deformity is not due to epiphyseal involvement but to malposition of the fragments, particularly in those cases with persistent ulnar displacement and rotation. In all but one of our cases in which such displacements were left unreduced, either gross varus deformity or loss of the carrying angle resulted.

It is to be remembered that the normal carrying angle of valgus is due to the normal obliquity of the distal end of the humerus. Any change in this plane will, of course, change the carrying angle. Displacement of the distal fragment ulnarly causes the articular surface to face medially and a position of varus is the result. (Figs. 1, 3 and 4.)



A B

C

D

FIG. 4. For descriptive legend see opposite page.

Normally the carrying angle is obliterated on pronation of the forearm. When the medial end of the distal fragment rotates posteriorly, the forearm follows the distal fragment, thus the distal fragment and forearm become, so as to speak, pronated on the proximal fragment. (Fig. 2.) By such rotation, the carrying angle becomes lessened or obliterated. In this respect it is interesting to note: (1) In two cases in which the ulnar displacement had been corrected but in which rotation persisted, a loss of the carrying angle was the result in each case. (2) In seven cases in which there had been persistent radial displacement and in which we might have expected an increase in the carrying angle, rotation has also been present which compensated for the radial displacement and normal carrying angles have resulted.

In order to prevent deformity it is, therefore, imperative to reduce ulnar displacement and rotation of the distal fragment, and to maintain such reduction. Maintenance of such a reduction we have found difficult with the accepted methods of treatment. After reduction and the application of either adhesive or a plaster cast, it is the customary practice to place the forearm across the chest. In so doing the forearm and the distal fragment are internally rotated and it is assumed, once reduction has been obtained, that the proximal fragment also undergoes such rotation. If the fragments are locked, especially in acute flexion, such rotation of the proximal fragment will occur. However, such flexion cannot always be obtained because of swelling or interference of the radial pulse. In the absence of such flexion, the obliquity of the fracture line and the narrowness of the humeral shaft at the fracture site render locking difficult. In this event, when the forearm is placed across the chest, only the distal fragment rotates and the position obtained by reduction is lost. With the recurrence of rotation, backward and ulnar displacement can easily occur, especially if the immobilizing agent fails to

FIG. 4. A, case showing original ulnar displacement in anteroposterior view. B, lateral view of same case. Note in attempting to get this view only the distal fragment rotated. The position of the shaft remains the same in both views. Similar rotation occurs, after reduction, in unlocked fractures when the forearm is placed across the chest. C, same case (anteroposterior view) two years later with opposite normal elbow. These views were taken with the condyles of both humeri lying in the same plane. The fracture has healed with almost 90 degrees rotation as in Figure 4B. Note views of condyles are anteroposterior while that of the shaft is lateral. D, same case, both elbows in lateral view. Note view of condyles is lateral while that of shaft is anteroposterior. The deformity in this case was largely due to this extreme rotation.

FIG. 5.

FIG. 6.

FIG. 7.

FIG. 5. Reduction by Cotton method of external rotation of the entire upper extremity with the forearm supinated.

FIG. 6. Cast applied to arm with elbow at right angles. This position is used only when swelling and interference of radial pulse allow no further flexion.

FIG. 7. Chest swathe of plaster applied holding distal fragment and forearm in external rotation. When further flexion is possible, such complete external rotation is usually not necessary.

give proper support for the weight of the forearm. We believe this practice of placing the forearm across the chest, to be the chief reason for our failures to maintain position.

We have been able to maintain position much more satisfactorily since the following method was adopted. Reduction is obtained by the method described by Cotton, namely, outward rotation of the entire arm. (Fig. 5.) By this procedure the elbow is flexed at a right angle and the forearm is abducted which tends to rotate the distal fragment externally. We also believe the forearm should be supinated. By so doing we are of the opinion that tension is placed on the pronator radii teres which further prevents the internal condyle from rotating backward. With the extremity thus rotated outward, the elbow is flexed as much as possible, depending upon the amount of swelling and the quality of the radial pulse. Plaster strips are then applied to the arm and allowed to set partially. A plaster swathe is then placed around the chest and incorporated in the plaster about the arm. (Figs. 6 and 7.) In this manner the fragments are locked in flexion and external rotation and the chances of recurrence are definitely less. After the adoption of this method, we found a similar procedure had been described by Eliason. He believed, in employing the acutely flexed position, that the hand should be carried to the shoulder of the injured side to prevent recurrence of rotation. He does not externally rotate the arm as we do, and in cases when acute flexion can be accomplished, his method should be satisfactory. When acute flexion cannot be maintained, further external rotation should be employed.

Some other interesting facts learned from this study are as follows:

There were two cases of so-called myositis ossificans. Both cases were slow in recovering function but all evidence of calcification and limitation of motion disappeared within two years from the date of injury.

Nerve involvement was seen in four cases. The radial nerve was involved in three instances, the ulnar in one. Both cases of anterior displacement showed nerve involvement, one radial and one ulnar. In all cases complete recovery occurred from six to ten weeks after injury.

There was but one case showing circulatory embarrassment. This was placed in traction for a few days and then reduced with an excellent end result.

Although the functional end results were excellent, some cases did show changes in the range of motion. In three cases there was a

loss of flexion of from ten to twenty degrees. Four cases showed a loss of extension of from ten to twenty degrees. In six instances there was an increase in the range of extension of from ten to twenty-five degrees without loss of flexion. Three of these cases have varus deformity.

In summarizing the end results of fifty cases of supracondylar fracture we find almost uniform excellent function. Cosmetically, there were twenty-seven excellent end results. There were two cases of increased carrying angle and three cases of decreased carrying angle. There were three cases of complete loss of the carrying angle. Although the above eight cases show change or loss in the carrying angle, they cannot be classified as showing poor cosmetic results. There were, however, fifteen cases of definite varus deformity which can only be classified as poor cosmetic results.

CONCLUSION

Supracondylar fractures with radial displacement and rotation of the distal fragment show excellent function and cosmetic end results, although complete reduction may not be obtained.

Varus deformity is due to ulnar displacement plus backward or internal rotation of the distal fragment. Persistence of either condition may lead to loss of the carrying angle or varus deformity. Persistence of both conditions leads almost invariably to the poor cosmetic result of cubitus varus.

Recurrence of deformity we believe is due to the practice of placing the forearm across the chest.

A method of immobilization is described which it is hoped will give better results.

There seems little excuse for poor cosmetic results in this type of fracture which we believe are to be universally found. The methods of reduction and immobilization were described by Cotton and Eliason eighteen years ago. The popular belief that accurate reduction is not essential to a good result is condemned.

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DISCUSSION

FRED H. SMITH (New York City): I would like to ask Dr. Aitken what he has been doing for the supracondylar fractures with marked circulatory disturbance in which the displacement was great and the soft tissue damage proportionately increased. In New York, we have been using the Kirschner wire and putting the arm in traction, hanging the elbow above the patient with the patient lying supine in bed. By that means, we are able to keep the elbow elevated. There is no obstruction to either the venous or lymphatic return, the patient is kept quiet and it is practically a painless procedure, and we are also able to control rotation of that distal fragment. If we find in subsequent check-up by *x*-ray that there is rotation, we can turn the forearm this way or that way and control that distal fragment.

Out of 218 cases in a nine-year period from 1929 to 1937, inclusive, we have used it in twenty cases, that is, just under 10 per cent, and out of that twenty, eight patients have had essentially perfect results. The remaining thirteen have had what would be called excellent results. A few of those had some loss of carrying angle and some loss of flexion. Some had one or the other, some had both, but the function was essentially perfect and they could do everything they wanted to.

As far as complications are concerned, we had three postoperative wristdrops, all of which cleared up subsequently. The only other complications were mild pressure sores at the side of the wire and no serious osteomyelitis.

Out of the entire series there was no case of Volkmann's paralysis. One patient came in with a beginning Volkmann's which had been going on only about five hours from the time of the onset. He had loss of pulse, some loss of nerve function and the fingers were cold. We put in the Kirschner wire and the impending Volkmann's immediately cleared up and he never developed any further complication.

ALEXANDER P. AITKEN (closing): I do not believe we have adopted any one procedure for the handling of these circulatory disturbances. In this one particular case, the arm was placed in traction with adhesive along the forearm and suspended from a Balkan frame. If there is any question of circulatory embarrassment, with an absent pulse, I know some men (and I have done it myself), have gone in and split the bicipital fascia. Other men have used Kirschner traction. We have not adopted any one method for handling it. It varies with the man who happens to get the case.

POSTOPERATIVE PAROTITIS WITH RADIATION TREATMENT

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PAROTITIS, when a complication of surgical procedures, is a very serious condition. Statistics gathered previous to irradiation therapy show a mortality rate of over 50 per cent. Blair and Padgett, in 1923, reported on thirteen cases of postoperative parotitis with seven deaths. Fortunately, it is a rather rare occurrence, the incidence being given as anywhere from one case in one thousand to one case in two thousand operations. Rankin and Palmer, in 1930, surveying a four year period at the Mayo Clinic, found twenty cases in 2,700 operations on the colon and rectum, a ratio of 1 to 135. They concluded there was an increased incidence with the increased development of surgery of the large bowel. They could not explain why. In 1937, Maurer reported thirty-three cases in 35,000 operations at the Munich Surgical Clinic. He also reported fourteen deaths in these thirty-three cases. I do not believe the high mortality rate of surgical mumps has been generally appreciated by the profession.

Numerous theories have been advanced to account for its occurrence, but none of them is fully satisfactory. In 1887, Stephen Paget investigated the records of one hundred cases and concluded that in those cases in which it followed operations on the genital organs it was due to close relationship between these organs and the parotid gland. Dr. Howard Kelley writes: "It is my conviction that postoperative parotitis is due to thumbing of the parotid gland by the anesthetist." It might be pointed out that the disease is not partial to operations upon the genital tract, and it has followed operations under local anesthesia in which there was no manipulation about the patient's jaw. Debilitation, dehydration and foci of infection in the oral and nasal cavities are generally mentioned as contributing factors, but great numbers of patients with these unfavorable conditions are operated upon daily and parotitis remains a rare complication. Moreover, as Rost points out, "It is almost never observed as a result of stomatitis."

Postoperative parotitis differs widely from simple parotitis or mumps. The latter condition, which is a virus disease, has a long

incubation period, no tendency to suppuration, and a negligible mortality; whereas surgical mumps has a short incubation period, does tend to suppurate, and has a high mortality. The theory that it was of embolic origin or due to pyemia has little support at present. Pyemic parotitis does occur in the presence of sepsis, but surgical mumps may occur in the absence of any apparent infection. In pyemic parotitis it has been shown that the infection begins around the vessels in the parotid gland, while in postoperative parotitis it begins around the ducts. Rost says, "There is no difference in the anatomical picture between the ascending and the hemotogenous routes of infection." The staphylococcus is the organism most frequently involved. This is true even though the patient may be suffering from an infection due to another bacteria.

The disease may follow any type of operation whether septic or not. I remember one fatal case that followed the dilating of a urethral stricture. It is usually ushered in with sudden high fever and malaise within a week after operation. The infected gland early shows swelling, and the diagnosis should be easily made. It is most important that the condition be recognized in its early stage, for a successful result demands early treatment. Nurses and interns should be instructed to be on the look out for the first signs or symptoms.

Many forms of therapy have been advocated in the past. Local applications, mouth washes, catheterization of Stenson's duct, use of vaccines, and wide incisions when suppuration is present, have been the general treatments.

Leithauser and Cantor treated twenty-three patients with large doses of Lugol solution. Twenty minims were given every three hours, day and night. Fulminating cases were given 1 to 2 dr. in 1,000 cc. of solution intravenously. Dosage was decreased as improvement took place. They advised aspiration with a large needle instead of incision if suppuration occurred. This treatment might be worth trying if radiation is not available.

The sulfonamides are probably of value. Walker and Allen report a case of bilateral postoperative parotitis treated with 10 gr. every six hours, but radiation was also used in their case. R. E. Smith reports quick recovery from a case of ordinary mumps under sulfanilamide therapy.

Bowing and Fricke reported on the use of radium in 184 cases at the Mayo Clinic. Thirty-two of these cases had bilateral involvement, and the mortality was 40 per cent. Of the other 152 patients, 19 per cent died. The authors added that 6 per cent died of parotitis

alone. They advised radium in moderate doses, with local treatment of warm, moist dressings, and incision when suppuration occurred.

In 1936, Dr. Fred M. Hodges, of Richmond, Virginia, reported that he had successfully treated a fairly large number of patients with acute, subacute and chronic parotitis since 1924 with roentgen therapy. He stated that the majority of surgeons were unaware of the value of irradiation in this field. He advised three to five treatments daily, or every other day, using 125 to 170 kilovolts, with about 125 to 200 R units. Dr. Hodges deserves the credit for bringing this form of treatment to the profession.

Following the method of Hodges, Latchmore and associates reported eleven cases of postoperative suppurative parotitis in which the patients were treated by *x*-ray therapy. Complete resolution occurred in six, two recovered after incision of a local abscess, and resolution was taking place in one when the patient died of another cause. Two patients treated late, that is, ten days after the swelling appeared, died.

Five cases of postoperative parotitis occurred on my service at the Bridgeport Hospital between May, 1940, and August, 1941. These patients were treated by radiation therapy under the direction of Doctors Parmelee and Lockhart, and the results are the reason for this report.

CASE REPORTS

CASE I. Mrs. M. L., age sixty-five, was operated upon May 27, 1940. A cyst of the right kidney was removed. On May 30, the patient complained of soreness in the left side of the face. The left parotid gland was swollen and tender; her temperature was 102°F. She was given 450 R units in the course of five days. The swollen gland subsided and the temperature was normal by June 9.

CASE II. Mrs. M. G., age eighty-five, was operated upon June 28, 1940, when a colostomy for intestinal obstruction was performed. On July 14, the left parotid gland was swollen and tender; the patient's temperature was elevated. *X*-ray treatment was started, and she was given 150 R units every other day for three doses. Her temperature was normal on July 22, and she went home on July 24.

CASE III. Mary F., age sixty-five, had a gastric resection for carcinoma on November 12, 1940. On November 16, the patient was listless and drowsy; her temperature was 101°F. At 8 A.M. it was noted there was a large swelling of the right parotid gland. At 9:30 A.M. she received her first radiation treatment of 195 R units. On November 17, the patient was given a second treatment, and two days later a third dose. The gland subsided

promptly and her temperature was normal by November 27. She was discharged one week later.

CASE IV. Mrs. E. W., age fifty-eight, had a cholecystectomy and freeing of adhesions performed on July 24, 1941, and also cecostomy for obstruction (inflammatory) of the colon. On August 4, the right parotid gland was swollen; her temperature was 103°F., and leucocyte count 33,000, with 91 per cent polymorphonuclears. *X*-ray treatment was started at once and repeated on August 5. The temperature dropped and the swelling of the parotid subsided in a few days. On August 14, there was a recurrence of the parotitis and *x*-ray treatments were again given on August 14, 15 and 16. The response was prompt and the patient proceeded toward a good recovery.

CASE V. Miss D. H., age twenty, was operated upon July 28, 1941, for acute purulent appendicitis. On July 31, there was swelling and tenderness of the left parotid gland. The patient had a chill and temperature of 103°F. *X*-ray treatment was started within a few hours after the swollen gland was first noticed. She received two treatments on successive days. By August 4, the temperature was normal and the parotid swelling much less. The patient had no further trouble.

I am unable to explain why these cases occurred. In only one instance were two of the patients in the hospital at the same time, and all were in different wards. Three had nitrous oxide ether anesthesia by machine. One had open drop ether after gas induction, and one local novocain infiltration. Different anesthetists were involved, as well as various doctors and nurses. There had been no similar cases for a long time previously, and there has been none since on my service.

Aside from radiation, local treatment only was employed in these cases. The radiation was begun early, and in no case did suppuration occur, although it appeared imminent in some of the glands. The prompt recoveries and absence of suppuration indicate that *x*-ray radiation is a very valuable agent for the treatment of postoperative parotitis, and we believe that the splendid results obtained in these cases were due to the early application of radiation.

As prophylactic measures, no elective surgery should be done when foci of infection are present in the nose, mouth or throat. Debilitated and dehydrated patients should receive proper preoperative treatment.

SUMMARY

1. Postoperative parotitis is a very serious complication.
2. *X*-ray radiation is a most effective treatment.

3. Radiation should be started early in the disease to prevent suppuration.

4. More attention should be paid to preoperative preparation of surgical patients.

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PERIPHERAL CIRCULATION IN RELATION TO TRAUMA*

WITH SPECIAL REFERENCE TO THROMBOSIS AND EMBOLISM

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WHEN I was first approached on the subject of peripheral circulation in relationship to injury, I had in mind that I was supposed to deal with injured blood vessels—the effect of healing of tissues distal to such lesions, etc. I have been informed, however, that Dr. Marble's idea was to have me discuss the question of thrombosis and embolism, which had interested us in this Hospital so much during the past year. I very much regret that one of your members, Dr. Henry H. Faxon, is not here to present this phase of the subject since he[1] and Dr. Claude E. Welch and Dr. C. E. McGahey have made a special study of this situation and have sent in for publication an excellent article on the subject. Since all of these men are now with the Military Forces, I will endeavor to give you a résume of our experience in this field.

Although thrombosis followed by embolism is not as common in the average patient who suffers an injury, as it is following many major surgical procedures, there are instances that make us realize that one must be on the alert for this possibility in all patients regardless of their type of illness or injury. For some time, it has been the policy of our *X*-ray Department to interpret certain signs in the chest as due to infarct with great accuracy. In addition to this, the Pathological Department have demonstrated that almost all fatal pulmonary emboli, which we have encountered, have originated in the region of the popliteal vein. When such a thrombus propagates in both directions, the long column of the femoral vein can produce an enormous clot and one that, when it breaks loose in its entirety, will very often completely occlude the pulmonary artery.

Several years ago, Dr. John Homans[2] of Boston read a paper before the New England Surgical Society advocating the ligation and division of the femoral or the iliac vein, following repeated sub-

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lethal infarcts in patients who had a low-grade inflammatory or mechanical condition in the vein producing thrombosis. Although this was an interesting observation that Dr. Homans reported, three or four cases at that time, believing that he had perhaps prevented fatal embolus under these circumstances, I doubt if there was any widespread attention paid to this suggestion. Actually, it was two or three years afterward before I found a case that I thought fitted into his group and on whom I was willing to do a high ligation. Gradually, there has come about a much more radical attitude about the entire situation.

First of all, one must get over the feeling that interruption of the femoral or even the iliac vein will produce severe invalidism. There occurs some swelling in the extremity after ligation but this is not disabling; it lasts for varying periods of time, but rarely handicaps the patient in his usual routine of life. The disability, in fact, is so slight that our internist colleagues believe that it is entirely minor as compared with the risk of waiting to see whether the patient would develop a more massive embolus. It only takes an instance or two in a man's experience of fatal embolus in an otherwise healthy patient to make him feel considerably more radical about this whole matter.

Fatal pulmonary embolus occurred in this Hospital prior to Dr. Lincoln Davis' study[3] of fifteen years ago at the rate of three deaths in every thousand patients who had been subjected to a major surgical operation.

After this, various methods of prevention of phlebitis were instituted, such as elevation of the foot of the bed, bicycle exercises of the legs, frequent turnings, etc. These methods, with perhaps better attention to the handling of tissue, the elimination of pressure bandages over large vessels, have (according to a recent study by Drs. Welch and Faxon[4] on patients in this same Institution) reduced the number of fatal emboli to about one in a thousand operations.

At first, it seems as though we might be able to pick up the early signs of phlebothrombosis or thrombophlebitis by a routine, bi-daily, careful inspection of the extremities of these postoperative cases. This is done by the resident staff religiously and frequently enough early signs can be noted, although these may be minimal. It is not at all uncommon now to have these signs picked up before the patient is aware of any disturbance or before the chart indicates that there is this complication impending. These examinations con-

sist in an observation of the extremities, both being uncovered; the question of whether the superficial veins are filled or empty; whether there is any swelling by actual measurements; whether there is any tenderness in the calf of the leg, and whether or not there is a positive Homans' sign.

This last sign is by far and above the most helpful of all and is determined by the following method: With the patient's leg in extension, the palm of the left hand is placed gently on the patella; the right hand grasps the distal segment of the foot, which is gently but forcibly dorsiflexed. If during this maneuver, the patient is perfectly comfortable, one can feel reasonably assured that the veins in the popliteal space are not involved in any pathological process. If, on the other hand, the patient complains of discomfort in the calf of the leg or in the popliteal space when this maneuver is done, one can then be reasonably sure that future developments will reveal a thrombosis in the vein. We do pay considerable attention to superficial phlebitis when it develops and believe that, when it is discovered, ligation at the foramen ovale of the long saphenous vein is indicated. This, we believe, has perhaps prevented a good many instances of deep phlebitis and has made it possible to shorten the convalescence of these patients tremendously.

Although we see a number of cases that certainly fall into the group which Dr. Alton Ochsner[5] has called phlebothrombosis, we have here in this community a larger number of true inflammatory thromboses of the vein than they seem to have in New Orleans. It is probably due to the fact that we have more lowered resistance due to our climate, etc., than they find in the deep South. It is a well known fact that we have more postoperative pulmonary complications in this region than they have in warmer areas. This we believe is due to the same difference in climactic conditions.

In the recent study, it was found that 95 per cent of all of the fatal emboli occurred in people who had absolutely no sign whatever of a previous thrombosis. They had not complained of any discomfort in their legs, they had no change in pulse, temperature, or respiration, and undoubtedly most of these should be classified as phlebothromboses. In these patients, the pathologist invariably found the origin of the embolus to be in the deep veins of the leg.

Further analysis showed that in the patients between twenty-five and forty-five years of age, there were very few deaths, although there were a good many infarcts in the lungs in this group. Before we began doing ligations, these were all treated conservatively and very

few of them died. In the older group beyond the age of forty-five, the number of fatal emboli was very much greater; the tendency increases as the patient grows older, so that we began ligating the femoral vein in the older age group with greater frequency and with more feeling of satisfaction than we now have when any patient of any age group has any indication of phlebothrombosis or thrombophlebitis, since such a patient is subjected to ligation without further ado.

It was thought that perhaps we could be intelligent and scientific about selecting these patients for vein ligations. Venography is now quite a well established method of outlining the veins and it gradually became used in the study of these patients. It was believed at the time that Drs. Faxon, Welch and McGahey sent their paper in for publication, that perhaps venography should be done pretty often in a doubtful or borderline case. Although venography is still done, I wish to say that we are not quite so sure that it is a completely reliable test and we know, with the *X*-ray Department shorthanded as they are, that it puts an extra burden on them which perhaps in the future we may usually avoid. The method of venography is well described in the article referred to above and when such a test is definitely positive, one is greatly assured that ligation is indicated. If, on the other hand, it is negative, one may feel with a clear conscience that the veins need not be ligated. The difficulty with the method is that in some instances the vein has appeared normal and yet the patient has had an embolus; and some of the veins that have been explored and ligated in the presence of a normal venogram have shown thrombosis in the vein. On the other hand, some of the venograms, that have shown no filling whatever in the deep veins, have misled us in that at operation the vein has been found completely free of any thrombus.

When multiple emboli have occurred, the patient may well develop considerable difficulty in his chest which continues even after vein ligation. In instances in which veins that have appeared to be normal have been ligated, the infarcts have continued always sublethal, leading one to assume that these infarcts may well have come from the neighborhood of a wound and would never have proved fatal under any circumstances. Such patients sometimes are helped by heparin, although our experience with heparin here has not been as spectacular as it has been in Toronto. So far we have had no experience with dicoumarin and this may eventually prove to be a tremendous help in this field.

There is one very definite feature that I think should be stressed and that is the ligation of both femoral veins rather than the one that appears to be involved. We have expected the misfortune of ligating the vein on the side that was obviously involved with thrombophlebitis only to have the patient succumb to fatal pulmonary embolus, and to find at autopsy that the thrombus that had produced the embolus had come from the supposed normal side. Therefore, there is some argument that if one ligates the obviously involved vein, one must then very seriously consider ligating the supposed normal side. This bilateral ligation is being carried out here now with more and more frequency.

The technic of the operation is extremely simple: Under local anesthesia guided by the pulsation of the femoral artery, one makes a 2½ to 3 inch incision over the course of the vein, starting in the crease of the groin. The long saphenous vein is investigated and if this is found not to be thrombosed and not varicosed, it is left alone. The femoral vein is exposed in the short space that is free below the profunda femoris. One can then pass two chromic catgut ligatures under the vein, leaving them untied but held by hemostats so that the bleeding can be controlled after the vein is opened. If one opens the vein transversely through half its diameter, leaving the posterior third or half of the vein intact, the manipulation is almost as easy as if the vein was completely divided and the control of bleeding from the vein, by holding up on the previously placed catgut ligatures, simplifies the procedure. After opening the vein, clots will exude from it and by using a glass-tipped Trendelenburg suction apparatus or a simple glass drinking tube, one can with suction pull the clot from the region of the iliac vein. After one has got all of this clot out, there should be free bleeding from the proximal segment. This bleeding is kept under control then by the elevation of the upper ligature. Then clot is milked and sucked from the distal segment of the vein, removing as much of it as possible. Frequently enough, clots from above and below put together may measure 16 or 18 inches in length. If one succeeds in getting the clot from the lower segment of the vein so that free bleeding takes place from below, the resulting edema from the ligation will be greatly diminished and the convalescence thereby will be shortened. After removing the clot from the vein, one simply ties the ligatures previously placed and then transfixes the vein with a cotton or silk suture distal to the catgut tie in order to insure closure of the vein in this region. One may leave the posterior half or third of the vein intact, thereby ligating the vein

more or less in continuity, or divide it. It seems to make no difference which method is adopted. We have reason to suppose that in time nature reëstablishes circulation through this tied-off vein, as we have venograms that would seem to prove that this actually did occur.

After ligation other things being equal as regards difficulty from the infarcts in the chest or the operative wound originally made, one may with perfect impunity make these patients ambulatory just as soon as their temperature is normal. It is a great comfort to be able to get these people out of bed at the end of forty-eight to seventy-two hours and have them walk about. They use at first an ace bandage on the lower extremity from the foot to the knee and often take this home with them but rarely use it for more than three or four weeks. The small amount of swelling which occurs after this time is so negligible and so unimportant from their standpoint that they prefer not to bother with the bandages. We have noted that there was some tendency for superficial veins to increase in size, temporarily at least, following the ligation of the femoral vein itself.

I am perfectly certain that time will finally settle the matter in a little more satisfactory fashion than it appears to be today. At the moment, I know we are ligating a great many veins that would not be the source of a fatal pulmonary embolism. I would suppose that perhaps we may save six lives out of each hundred patients on whom we do vein ligations. This is a definitely worth while salvage but with more experience and a more careful analysis, perhaps more useful standards of indications, we may be able to select these patients better and reduce the number of ligations with a consequently higher ratio of rescues. At the moment, it is difficult to persuade our medical colleagues that every infarct of the chest should not have immediate bilateral femoral ligation. We are at a loss to know how to answer this other than to comply with their request because, in the first place, we must admit that this is not a serious operation; it does not produce a serious handicap to the patient afterward; and, of course, at the moment we have no way of telling whether or not that particular patient would develop a massive embolus if his veins were left intact. There are many problems to be settled and one, I am sure, is how can we tell whether the infarct has not come from the heart, from the operative site, or from some smaller vein in the body other than the ones that are large enough to produce the amount of thrombus necessary to occlude the pulmonary artery completely. We believe, in time, that many of these questions will be solved; we plan a further analysis of our experience which is now getting greater

day by day. I think it is probably safe to say that at this time we have ligated already in this Hospital approximately 150 veins and this would probably represent about 125 patients. At the rate we are going, it will not be long before our experience will be very large. We know that it is an operation that can be done simply and easily; it takes almost nothing out of a very ill patient. It requires twenty to thirty minutes of time for each side, and the results in some instances are so spectacular that we believe it well worth while. In addition to the prevention of fatal embolus in the cases of thrombophlebitis, we find that the swelling and the pain resolves very much more quickly following the opening of the vein, the removal of the clot, and the ligation of the vein, than it would under ordinary circumstances. Patients, who used to remain in bed for six weeks to recover from a thrombophlebitis, would often be able to leave the hospital under these circumstances within a week.

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QUESTIONS AND ANSWERS

E. Payne Palmer (Phoenix, Arizona): I should like to ask Dr. Allen if he has had to go into the iliac vein to ligate in any of his cases. We have had to do so in two and found the clot rather extensive when we opened up the femoral. Therefore, we made the Cameron incision, pushed the curtain in back and went back of the clot into the iliac.

Dr. Allen: I know a doctor who has done that several times, and we have supposed that we might have to do likewise. He used that method of removal of the clot, but if you get the upper segment of the clot out, I think you need not fear fatal embolism. Any thrombus remaining above the ligature would probably stay there and not go to the lungs.

Dr. Palmer: How do you know you are going to get it all out?

Dr. Allen: I do not think it matters. You may get a small infarct as we did once in thirty-nine cases, but it is not fatal. What you are trying to avoid is enough clot going to the pulmonary artery to cause death, and this does it. Incidentally, interrupting the flow in the vein is very effectively done by the ligation of the femoral vein at this point.

Dr. Darrach asked me why an inverse incision instead of a longitudinal one. You have not much room unless you tie off small veins that are going into the muscles. There is about a centimeter between the profunda and muscle branches, which is enough if you use a transverse incision.

FREDERIC W. BANCROFT (New York City): Might I ask Dr. Allen what result he had had from blocking the sympathetic?

DR. ALLEN: We have not had as good luck with it as they have had in New Orleans. I think we have a different kind of thrombophlebitis here on the East Coast. I mean that, I have been down there and seen these cases and know they do have excellent results with this method, but our phlebitis, once it starts, is a difficult one to control. It takes six weeks to get over it; however, we have used this method very extensively and we think it has helped a great deal. It is one of the methods we would use if we were not going to ligate. Patients object to having their backs stuck too many days in succession and their phlebitis lasts too long here to make us believe we should depend upon that method alone.

KELLOGG SPEED (Chicago, Illinois): Have you used Dicoumarin at all?

DR. ALLEN: I have had absolutely no experience with it. I do not know whether that is going to be effective or not, but I hope so.

DR. SPEED: It is very effective, at least in cases I have seen.

FRASER B. GURD (Montreal, Canada): We have used Dicoumarin for the last eight or nine months, and we are of the opinion that it is useful, and as compared with heparin it costs only five cents a day.

DR. ALLEN: You say it is useful?

DR. GURD: As compared with heparin it seems to be as useful. It is given by mouth, and two or three days' administration lasts for about two weeks. The only objection that we can see is that you cannot get your clotting time back once you have compressed it, as compared with heparin.

DR. ALLEN: I do not think we have used it in this hospital as yet.

DR. GURD: I do not think it is on the market.

DR. SPEED: It is only being given out experimentally.

WILLIAM L. ESTES, JR. (Bethlehem, Pa.): How long have you kept the patients inactive after ligation of the femoral vein?

DR. ALLEN: Everything else considered, forty-eight hours is enough, unless there are other reasons for a longer period. If the patient is febrile still from his infarction, one should naturally keep him quiet longer, but forty-eight hours is sufficient in the case of the ordinary patient. For instance, the one upon whom I think I did the unnecessary ligation, I got out of bed the next day.

WILLIAM DARRACH (New York, N. Y.): How much do they swell up afterward?

DR. ALLEN: Very little if you ligate them below the profunda.

G. GAVIN MILLER (Montreal, Canada): Dr. Allen said he had had no deaths following ligation. I should like to report one. We had a patient with

an infarct; we ligated, and a few days later he died of embolism. We also had a death following ligation within six days.

DR. ALLEN: Were you able to get an autopsy on the patient that died? Do you know for certain that the fatal part of the embolus came from the side you ligated?

DR. MILLER: No.

DR. ALLEN: We have been pretty lucky along that line, because I am perfectly certain that patients are going to die from the side that we have not ligated. This fear has made us more willing to do bilateral ligations.

H. GURTH PRETTY (Montreal, Canada): Do you find any increase in the number of these cases as the result of using cyclopropane?

DR. ALLEN: Cyclopropane has not been used in this hospital since the fatal explosion that occurred in Boston. The trustees did the same thing about ethylene. We had three or four minor explosions with that and they said, "You cannot have it any more," just about the time we had become fond of it; and the same thing happened with cyclopropane. We have not used any for a number of years. I had not realized it would increase this condition.

DR. PRETTY: In looking up a great many cases of pulmonary emboli, we found that the great majority of cases in various hospitals occurred following spinal anesthesia; next in order were those following cyclopropane, and next in order those following open ether, on account of the slowing of the blood stream postoperatively.

DR. ALLEN: I cannot quote you the exact figures, but when Faxon looked over our fatal cases of emboli prior to the beginning of this recent work, he sorted them out as to the kind of anesthetic agent that had been used and came to the conclusion that here, at least, we could not see that it made any difference. The greatest percentage from any department came from the urological service, probably because they were dealing with a much older age group.

FRACTURES OF THE METACARPALS EXCLUSIVE OF THE THUMB*

A NEW METHOD OF TREATMENT

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FRACTURES of the metacarpals, although commonly regarded as trivial, are actually serious injuries frequently resulting in cripping deformities due to faulty union and contractures of the finger joints. This is largely a result of the difficulty in reducing the fracture and maintaining the proper position. Other factors are the lack of interest and the less consideration generally given to the early treatment and immobilization of these fractures than that given to fractures of the larger long bones.

It is admitted that simple splinting is quite satisfactory in fractures of the metacarpals without displacement. On the other hand we are convinced that the deformities (Fig. 1) and disabilities which follow metacarpal fractures with displacement are due to the failure to properly reduce the fractures by the usual methods and the inability or failure to maintain the reposition (restoration of bone length and prominence of the knuckle). The latter is influenced no doubt by the endeavor to prevent loss of joint function by too early mobilization of the finger joints.

Prior to 1932, practically all metacarpal fractures were treated by simple bandaging over a roller bandage with little or no attempt made to correct the displacement.

In 1932, McNealy and Lichtenstein[1] pointed out the unsoundness of the roller bandage method of treatment and called attention to the mechanics involved in these fractures and advocated treatment with a straight dorsal splint with the hand in extension. Since then the trend has been to immobilize these fractures in extension, frequently using traction, with either a straight dorsal or a banjo splint. This form of treatment has been recommended by Scudder,[2] Key and Conwell,[3] Magnuson,[4] Christopher[5] and others. In this connection Cotton[6] stated "there is no longer any excuse for treatment with the hand flexed either on the once much used roller bandage or the more

* From the Surgical Service of U. S. Marine Hospital, Boston

modern aluminum substitute." Owen[7] also condemned the use of "a roller bandage as a panacea for fracture of metacarpals."

Although the so-called extension treatment with or without the

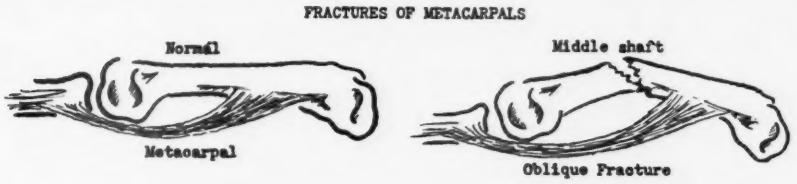


FIG. 1.

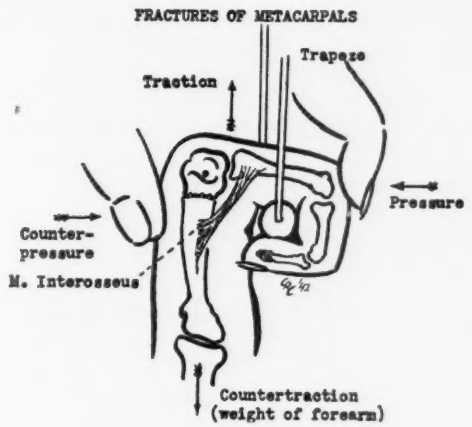


FIG. 2.

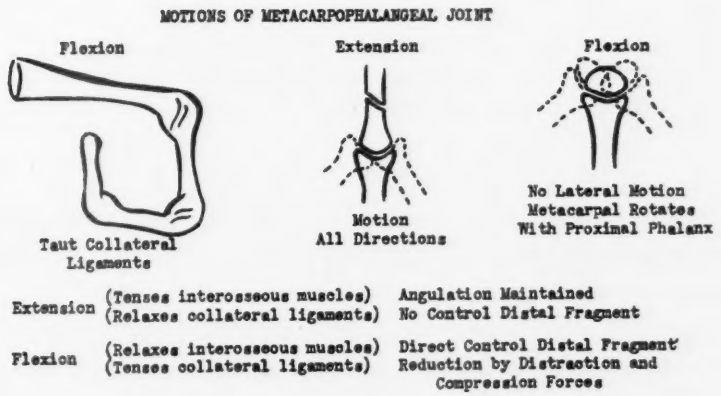


FIG. 3.

FIGS. 1–3.

use of traction indicated progress in the treatment of fractures of the hand, joint dysfunction due to capsular contractures of the fingers

frequently occurred after this form of treatment. Koch[8] states that fixation of the finger joints are prone to occur with the metacarpophalangeal joints immobilized in extension or with the proximal

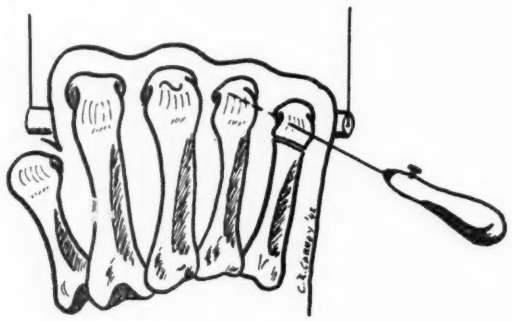


FIG. 4.

interphalangeal joints immobilized in flexion. To prevent these contractures Koch advocates immobilization of the hand in the optimum position for function, i.e., position of grasp.

Further progress was made when Jahss,[9] in 1938, reported a new method of treatment for the reduction of these fractures based on the

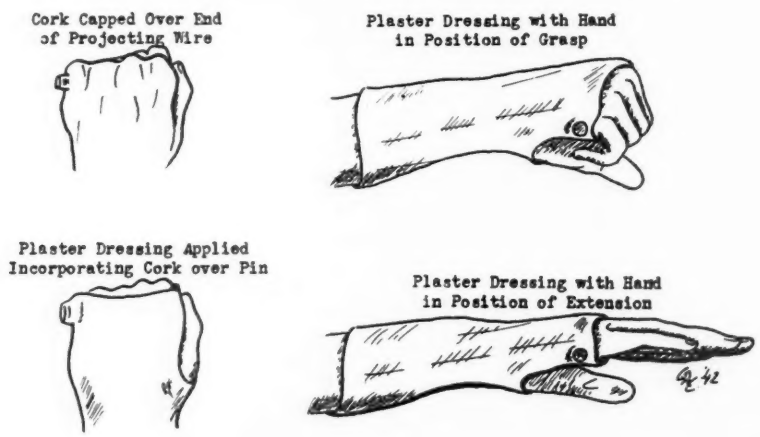

Cork Capped Over End of Projecting Wire
Plaster Dressing with Hand in Position of Grasp
Plaster Dressing Applied Incorporating Cork over Pin
Plaster Dressing with Hand in Position of Extension

FIG. 5.

correction of the anatomical deformity with the metacarpophalangeal and interphalangeal joints in flexion, and also with immobilization of the affected fingers in flexion.

As indicated by Jahss we have found that the reduction of the deformity is facilitated with the metacarpophalangeal and proximal interphalangeal joints of the involved finger held at 90 degrees. Unfortunately, however, plaster immobilization of the fingers in flexion usually results in limitation of extension power of the fingers.

Such contractures frequently require intensive physiotherapy for a considerable period after the removal of the cast. In an endeavor to correct the anatomic deformity and at the same time to prevent the restriction of finger motions which follow the immobilization of the finger joints we present a new method of treatment for these fractures.

TABLE I

AGE RANGE AND METACARPAL INCIDENCE

Ages: 15 to 47
Hand: Left 5; Right 17
Metacarpal: Single 20; Multiple 2
(Both multiple fractures were of 4th and 5th)
5th Metacarpal—15 3rd Metacarpal—4
4th " — 3 2nd " —2

TABLE II

FRACTURES OF METACARPALS ACCORDING TO CLASS OF WORKER AND MODE OF INJURY

Class of Worker	Mode of Injury				Total
	Fight	Falling or Moving Objects	Fall	Struck or Jammed Hand	
Industrial	0	3	3	6	12
Seamen	3	1	1	2	7
CCC	1	0	1	0	2
Schoolboy	0	1	0	0	1
Totals	4	5	5	8	22

TABLE III

METHODS AND RESULTS OF TREATMENT

Methods of Treatment:
Closed treatment—16 Open treatment—6
Indications for the Open Operations:
Delayed union—1 Compound fracture—1
Faulty union —3 Fracture-dislocation—1
Results (based on function and absence of deformity):
Excellent 14 Satisfactory 7 Poor 1

The method of treatment has been used in twenty-two cases of fractures of the four inner metacarpals, including both shaft and so-called "knuckle" fractures. The method is believed to be superior to the older methods of treatment.

The basis of treatment, as indicated above, is the early correction of the primary anatomic deformity of the fracture and the rigid fixation of the fragments, with the hand in the position of grasp (opti-

A

B

FIG. 6. Roentgenogram showing fracture of distal end of fifth metacarpal; A, before reduction and B, after reduction with fixation by pin and plaster.

FIG. 7. Roentgenogram showing fracture of middle shaft of fifth metacarpal; A, before treatment; and B, after treatment.

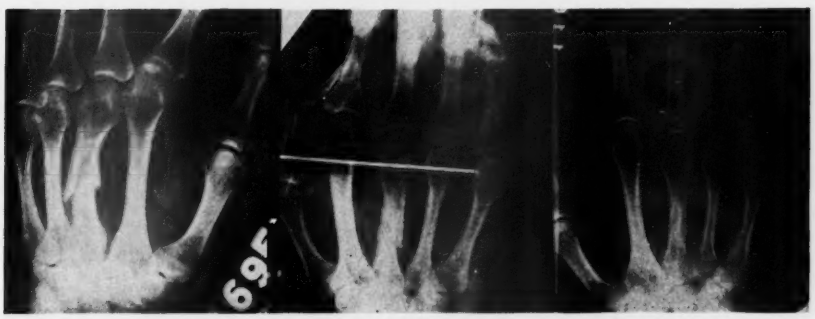
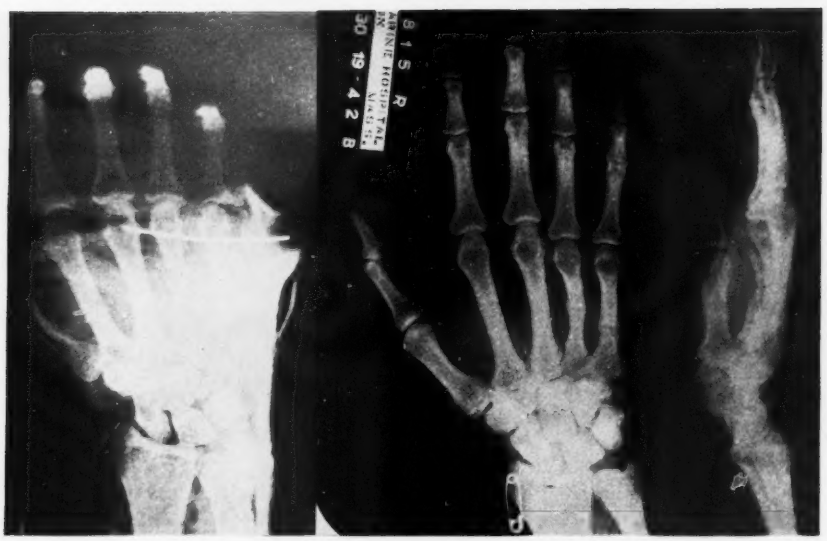


FIG. 8. Roentgenogram of fracture followed by delayed union, third metacarpal; A, ten weeks after injury; B, after open operation (intramedullary bone graft and fixation by pin and plaster); and C, one year after open operation (good anatomical and functional result).

A



B C

FIG. 9. Roentgenogram showing fractures of fourth and fifth metacarpals; A, before reduction; B, after reduction and fixation by pin and plaster; and C, six weeks after reduction.

mum function), but with all metacarpophalangeal and interphalangeal joints free during the period of immobilization.

The reduction of the fracture is accomplished by closed methods. The forearm is suspended vertically with the hand grasping a small trapeze. (Fig. 2.) The phalanges flexed over the small cylinder of the trapeze produces the traction and the weight of the forearm and arm the countertraction forces. The flexed proximal phalanx relaxes the interosseous muscles and tightens the collateral ligaments (Fig. 3) and synovia (capsule) of the metacarpophalangeal joint, thus allowing positive control of the distal fragment of the fractured metacarpal. The actual reduction of the fracture is effected by compression by means of the surgeon's left index finger and thumb. (Fig. 2.) The pressure is applied over the flexed proximal interphalangeal joint, and the counterpressure over the dorsum of the hand (the site of the dorsally bowed metacarpal fragments).

With the fracture reduced by the distraction and compression forces, rigid fixation is secured by means of a short Kirschner or Mathews wire about 2½ inches in length inserted transversely as a "pinion" through the distal ends (heads) of the fractured and adjacent one or two metacarpals. (Fig. 4.) Novocain solution is used as a local anesthetic necessary at the site of the insertion of the wire and between the heads of the fractured and adjacent metacarpals.

Finally, a very light plaster of Paris or starch bandage, incorporating a small cork capped over the ½ inch of wire protruding through the skin (the site of insertion) is applied to the hand and distal half of the forearm. The fingers are not encased in the plaster or starch dressing but are free permitting motion of the metacarpophalangeal and interphalangeal joints. (Fig. 5.) The continued free motion prevents capsular contractures of the finger joints so frequent after the usual methods of immobilization, particularly after immobilization in extension. The immobilization (wire fixation with plaster or starch bandage dressing) is continued for at least three weeks.

Usually after a week of physical therapy in the form of whirlpool and increasing active use of the hand, the patient can return to his usual work.

SUMMARY*

1. The seriousness of metacarpal fractures, commonly regarded as trivial injuries but frequently resulting in crippling deformities is

* Since the presentation of this paper, the authors have treated ten additional pa-

emphasized. These disabilities far in excess of the apparent importance of the fracture are due, to a considerable extent, to the lack of interest and the less consideration generally given to the early treatment and immobilization of these fractures than that given to fractures of the larger bones.

2. The difficulties of treatment with special reference to past progress made in the treatment of these fractures are briefly reviewed and discussed.

3. A new method of treatment used in twenty-two cases of fractures of the four inner metacarpals including both shaft and the so-called knuckle fractures is presented. This method is believed to be superior to the older methods of treatment. The basis of treatment is the early correction of the primary anatomic deformity and the rigid fixation of the fragments, with the hand in the position of grasp (optimum function) but with all metacarpophalangeal and interphalangeal joints free during the period of immobilization.

4. The results of treatment are shown in the accompanying tables and representative roentgenograms.

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tients. In these cases, the actual reduction was accomplished solely by manipulation (compression forces with the fingers in flexion) without the use of the trapeze and using intravenous pentothal-sodium for anesthesia. The results were excellent in all cases. The authors, therefore, recommend this modification as the method of choice for the reduction of these fractures but with the same wire fixation and plaster dressing as described in their original manuscript.

EXPERIENCE WITH BURNS AT THE HOSPITAL FOR SICK CHILDREN

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DUE to the limited space, it was decided that rather than stimulate any debate concerning the relative merits of different forms of therapy, to tell the realities as we have found them. With minor exceptions my ideas on results will not be presented, as they would add little to the already voluminous writings on the subject. Certain events concerning burns, however, appear to have been largely overlooked or to lack emphasis in the literature. It is chiefly to these that attention will be drawn. Thus this paper deals principally with facts about cases treated as in-patients from 1913 to 1942, all of these since 1932 being looked after by the author.

Nineteen-thirteen was picked, as from this date suitable records have been kept and are available. The cases which number over 1,800 are divided into four periods. In August, 1925, tannic acid was used first, so that this represents the end of the first period. Statistics until 1928 were gathered by Dr. R. I. Harris, who was then on the hospital staff. Thus this time has been taken as the end of the second interval. The third goes to the end of 1937, when sulfonamides were first used in our cases of burns, and the last is until the end of 1941.

The table (Table 1) by which the area of surface affected was deduced is shown. This is practically the same as Berkow's table. Allowances were made for discrepancies in this due to dealing with children of different ages. Until 1928, Dr. Harris made rough measurements of the areas and used no specific chart.

The second table shows the mortality statistics for the period 1913 to 1925. The causes for death have been divided into shock, toxemia and sepsis. These terms as used here must be defined. Listed under shock are those who died within ten hours of the accident, provided they did not show the signs ascribed to the next stage. The shock as herein described is what is called primary shock. It is my opinion that the term primary shock as applied to burns might well be dropped, and this column be added to the next. The shortest time in this whole series between accident and death was two hours, which is plenty for the development of the general reaction to a severe burn.

The term, "toxemia," as used here is referred to more commonly as secondary shock, or occasionally secondary shock and toxemia. Because of my belief that toxins cause the "oligemic anoxia" called

TABLE I

PERCENTAGE OF TOTAL BODY SURFACE OF COMPONENT PARTS
AS USED AT THE HOSPITAL FOR SICK CHILDREN

HEAD	6%						
NECK	2%						
BODY	36%	FRONT 12	BACK 12	ONE SIDE	7	(X2)	
UPP R LIMB	16%	ONE ARM 3½	ONE FOREARM 2½	ONE HAND	2	(X2)	
LEGS	30%	ONE THIGH 11	ONE LEG 6	ONE FOOT	2	(X2)	

secondary shock, as well as the other findings in this stage, all patients dying with these features have been listed under the one heading. This group of signs and symptoms are easily noted in burns of moderate or severe degree. They include an elevated temperature. This is usually about 101° to 102°F. and varies somewhat depending upon a number of factors, such as the extent of burn, the size of patient, etc. There is a fast pulse rate out of proportion to the temperature. Next, there is drowsiness, which may go on to coma and death. This drowsiness is occasionally disturbed by periods of irritability. Often there are localized twitchings and sometimes generalized convulsions. Vomiting commences early in severe burns. It may become continuous and in appearance is like coffee grounds. There is bright red blood occasionally. The limbs become moist and cold. One can blanch a grayish blue area on a limb and the return of color is very slow. The blood pressure falls eventually. The urine is of small volume and has a high specific gravity. Albumin may appear in severe cases as well as hyaline and granular casts. Acetone may be present.

Death while the above mentioned clinical features are manifest, has been listed under toxemia in this classification. The earliest was about four hours after the accident, although most occurred in three to four days, while a small number of cases were as late or later than ten days. If the blood is examined, it is found that changes have happened before the clinical findings are noted, great emphasis being placed in recent years on the hemoconcentration. While these changes occur before the clinical signs and symptoms, they may be largely or completely corrected before the patient dies. Under the column of sepsis are listed the cases who had a swinging type of fever and discharge from the burn area. The blood culture may or may not have been positive.

During the period 1913 to 1924 (Table II) the average mortality rate was 35.2 per cent. The shock and toxemia columns take in most of the deaths. The yearly admissions rose during this time to nearly

TABLE II

YEAR	TOTAL BURNS	DEATHS	SHOCK	TOXAEMIA	SEPSIS
1913	11	6(55%)		6	
1914	18	7(39%)	2	4	1
1915	22	8(36.4%)	5	3	
1916	37	14(37.8%)	2	8	4
1917	47	18(38.3%)	7	9	2
1918	29	10(34.5%)	6	1	3
1919	35	11(31.4%)	2	8	1
1920	38	13(34.2%)	3	7	3
1921	35	13(37%)	2	7	4
1922	46	13(28.3%)	3	8	2
1923	48	10(20.8%)	1	7	2
1924	46	14(30.4%)	5	5	4

fifty. The local treatment varied between vaseline, picric acid, ambrine and sodium bicarbonate. The general therapy included infusions of saline, calcium chloride and calcium lactate. The first transfusion was performed in 1919. The first exsanguination transfusion was in 1920. The average survival time excluding deaths after ten days was 1.7 days. A large number died in the first twenty-four hours.

TABLE III

YEAR	TOTAL BURNS	DEATHS	SHOCK	TOXAEMIA	SEPSIS
1925	60	10(16.6%)	1	5	4
1926	52	9(17.3%)	2	2	5
1927	68	10(14.7%)	1	4	5
1928	50	8(16%)	1	4	3

The next table (Table III) takes the same statistics from 1925 to 1928 inclusive. The average number of admissions increased a little. The mortality rate dropped suddenly to approximately one-half of those previously recorded, now being 16.1 per cent. The first column claimed fewer lives. The percentage of deaths due to sepsis when compared to the addition of the other two columns rose. In this period local treatment was tannic acid. General treatment included whole blood transfusions, exsanguination transfusions and intravenous electrolytes. The average survival time (excluding death after ten days) was 3.4 days, which represents a considerable improvement over that of the previous period.

Table IV takes the data until 1937. The average number of admissions increased to eighty-five. The mortality rate dropped still further to 11.8 per cent. Toxemia claimed most lives. Sepsis still

TABLE IV

YEAR	TOTAL BURNS	DEATHS	SHOCK	TOXAEMIA	SEPSIS	OTHER CAUSES
1929	72	7(9.7%)	1	2	2	2
1930	87	14(16.1%)	2	8	3	1
1931	84	8(9.5%)	1	7	0	0
1932	90	16(17.7%)	0	9	2	5
1933	116	12(10.3%)	0	8	4	0
1934	62	7(11.3%)	0	5	1	1
1935	106	6(5.7%)	1	4	1	0
1936	80	11(13.8%)	0	4	4	3
1937	85	10(11.8%)	1	9	0	0

played a part but this was less serious. A new column was introduced, because in a children's hospital due to intercurrent infections, it was impossible to ascribe all the deaths to the foregoing three columns.

TABLE V

BURNS TREATED WITH FERROUS OR FERRIC CHLORIDE

NUMBER		EXTENT
56090	621 B	Less than 5%
60058	672 A	5-10%
67076	623 A	5%
66986	673 B	Less than 5%
71938	624 A	5-10%
81884	651 B	5-10%
82110	652 B	5%
82223	655 A	5%
83364	661 A	30%
83579	676 B	5-10%
83660	665 A	5%
83678	666 A	10%
83791	666 B	5%
83793	668 A	5-7%
83908	668 B	5%
83934	669 A	5-10%
84055	669 B	5%
84088	670 A	5%
84397	678 A	5-10%
84611	679 A	Less than 5%
84640	679 B	15%
84720	680 A	5-10%
84723	680 B	35%-----Death
84851	681 A	5-10%
84922	681 B	Less than 5%

One death (35%) burn - considerable trouble due to infection - toxaemia noted in some cases but extent of burn not great except in one case of sunburn.

The local treatment still remained mainly tannic acid. Whenever any new idea seemed to merit, it was tried on a selected number of cases. For example Table v shows the twenty-five patients on whom ferric and ferrous chloride solutions were used. The general treatment still consisted of blood transfusions and intravenous electrolytes in the early stages. Exsanguination transfusions were discontinued, the last being performed in 1930. The survival time (excluding deaths after

ten days) was 3.8 days, which represents a slight improvement over the preceding period.

Table VI shows the period from 1938 to 1941 inclusive. The

TABLE VI

YEAR	TOTAL BURNS	DEATHS	SHOCK	TOXAEMIA	SEPSIS	OTHER CAUSES
1938	100	4(4.0%)	0	2	1	1
1939	58	1(1.7%)	0	1	0	0
1940	108	1(.9%)	0	1	0	0
1941	107	5(4.7%)	0	5	0	0

TABLE VII

MORTALITY RATES

1913 - 1924	35.2%
1925 - 1928	16.1%
1929 - 1937	11.8%
1938 - 1941	2.9%

TABLE VIII

AVERAGE SURVIVAL TIME

EXCLUDING DEATH AFTER 10 DAYS

1913 to 1924 inclusive	1.7 days
1925 to 1928 "	3.4 days
1929 to 1937 "	3.8 days
1938 to 1942 "	5.0 days

average number of admissions except for a small year in 1939 was over 100. The mortality rate for this period was 2.9 per cent, which is a marked drop. The deaths are practically all due to toxemia, there being none from sepsis in the last three years. The average survival time (there were no deaths after ten days) was five days. The early local treatment in this period was chiefly tannic acid. Sulfonamides were used locally and generally in the later stages. General treatment in the early stages consisted of blood transfusions, normal and concentrated serum and intravenous electrolytes. Cortin has been used since 1939 in severe cases. Table VII compares the mortality rates in the periods described and Table VIII gives the average survival time (deaths after ten days excluded). While the ten-day exclusion date was chosen because of the prevalence of contamination or infection in deep burns after the lapse of such a period, we have had patients die after this time with what we have listed as toxemia, as there was

no clinical evidence of infection. The toxemia of infection, when added to the severe illness of the early stage, is more likely to cause death, when either factor alone may not produce such an end.

TABLE IX

BURNS

MORTALITY ARRANGED ACCORDING TO THE EXTENT OF THE BURN 1913-1928

	NUMBER	DEATHS	MORTALITY
10% or less of total skin surface	391	30	7.7%
10 to 20%	134	52	38.8%
20 to 30%	34	23	67.6%
30 to 40%	35	24	68.6%
40 to 50%	16	16	100.%
50 to 60%	11	11	100.%
60 to 70%	7	7	100.%
70 to 80%	11	11	100.%
80 to 90%	3	3	100.%
	642	177	27.5%

TABLE X

BURNS

MORTALITY ARRANGED ACCORDING TO THE EXTENT OF THE BURN (1929-1937)

	NUMBER	DEATHS	MORTALITY
5% or less	473	11	2.3%
5-10%	174	8	4.6%
10-20%	65	20	30.8%
20-30%	39	26	66.6%
30-40%	11	7	63.7%
40-50%	8	7	87.5%
50-60%	4	4	100. %
60-70%	4	4	100. %
70-80%	1	1	100. %
80-90%	2	2	100. %
90-100%	1	1	100. %
	782	91	11.8%

The cases in the four periods as previously outlined were then investigated as to the mortality rates depending on the extent of the burn. The first two periods are put together in Table IX. It is to be noted that no cases with over 40 per cent of the surface area affected lived. The mortality rate rose very rapidly after 10 per cent and approximately one in three patients between the 10 and 20 per cent areas died.

Table x takes the same statistics until the end of 1937. Here the cases were further broken up into area of 5 per cent or less, and areas between 5 to 10 per cent. It is seen that there is practically no change

TABLE XI

BURNS

MORTALITY ARRANGED ACCORDING TO THE EXTENT OF THE BURN (1938-1941)

	NUMBER	DEATHS	MORTALITY
5% or less	247	I	0.4%
5-10%	81	I	1.2%
10-20%	32	I	3.3%
20-30%	6	2	33.3%
30-40%	3	2	66.6%
40-50%	2	2	100. %
50-60%	2	2	100. %
60-70%	0	0	100. %
70-80%	0	0	100. %
80-90%	0	0	100. %
90-100%	0	0	100. %
	373	II	2.9%

between these figures and those of the preceding table, which mainly represented forms of therapy other than tannic acid. It must be remembered, however, that the former table concerned the cases until 1928. The figures for surface area were obtained by Dr. R. I. Harris, and it is my opinion from review of the charts of these cases that there was some error of estimation on the high side. Thus a truer comparison between this and the preceding table is that of total mortality rates, which it is seen have fallen greatly. In the statistics since 1928 erythema was not included in the estimation of the surface affected.

Table xi takes the same statistics until the end of 1941. While the total number is approximately one-half that of the two preceding tables, the mortality rates have fallen markedly in the 10 to 20 per cent groups, and the 20 to 30 per cent groups, where it is the rule rather than the exception now to have the patient live.

Table xii analyzes the reasons for death in the burns of 5 per cent or less from 1929 to 1942. Sepsis and intercurrent infection have been the main causes. Toxemia has been listed in two cases in which there is a possibility for difference of opinion. This explains the query after each case. The main thought to be derived from the foregoing is that burns under 5 per cent in area are not serious from the standpoint of secondary shock or toxemia.

Many of the mortalities were followed by postmortem examination. The findings were remarkably uniform depending upon the period between accident and death. From the examination of reports

TABLE XII

BURNS

PERCENTAGE INCIDENCE OF BURNS OF 5 % OR LESS IN EXTENT BY YEARS

			MORTALITY % FOR YEAR	MORTALITY FOR 5% BURNS
1929	55	76.3%	9.7%	3 (1 sepsis) (2 intercurrent)
1930	59	63.2%	16.1%	3 (1 toxaemia?) (1 sepsis) (1 intercurrent)
1931	50	59.5%	9.5%	0
1932	40	44.4%	17.7%	1 (intercurrent)
1933	66	56.9%	10.3%	1 (sepsis)
1934	39	62.9%	11.3%	1 (sepsis)
1935	70	66 %	5.7%	0
1936	54	67.5%	13.8%	2 (2 intercurrent)
1937	43	50.5%	11.8%	0
1938	68	68.0%	4.0%	1 (toxaemia ?)
1939	31	53.5%	1.7%	0
1940	80	74.1%	0.8%	0
1941	68	63.5%	4.7%	0

TABLE XIII

CORTIN 14 CASES

HISTORY NUMBER	TIME OF DEATH(DAYS)	EXTENT OF BURN	AGE IN YEARS
97951	4	46%	8
110888	2	55%	3
114153		Less than 10%	1
115710	7	20%	3
116368	2	40%	1
10625	-	30%	6
115751	-	10%	3
95280	-	25%	11
95740	4	50%	2
118082	3	40%	9 mos.
120152	-	10%	4
120554	3	30%	9
120663	5	25%	2
121997	-	25%	3

14 cases---8 deaths---average extent 38.2---average survival time 3.6 days

6 survivals---average extent 18.3%

of fifty-six autopsies between 1920 and 1942, the following items were found:

Following death in the first twenty-four hours (twelve cases) there was mainly congestion of viscera, with petechial hemorrhages in the organs and connective tissues. This was most noticeable in liver, lungs, spleen, lymph glands, kidneys and adrenals, and in the

subpleural and subpericardial connective tissues. The head was not often included in the examination, but when it was, the same condition obtained here. Congestion in the gastrointestinal tract was not prominent at this time. It seemed to begin in the first part of the duodenum with more widespread involvement later. Rarely there were beginning fatty changes in the liver, and in one case there was some degeneration in the tubules of the kidneys.

Following death in the forty-eight-hour period (nine cases) the preceding changes were more accentuated. The congestion was more marked, petechiae were widespread and perhaps larger. Edema made its appearance. The liver changes were more advanced, with central liver necrosis. Two cases showed nephrosis. There were cases of congestion and erosion (not deep ulceration) of the mucous membrane in areas in the esophagus, stomach and duodenum. The lymph glands were edematous, large, and congested, and showed petechial hemorrhages. This included the regional as well as the mesenteric and mediastinal glands. Quite often the term, acute mesenteric lymphadenitis, was used in the description. In one microscopic report it was noted that there was an increase in reticulo-endothelial cells and some of these showed phagocytosis for broken down red blood cells.

In death in three days (seven cases) liver damage was the most striking feature. There was extreme fatty degeneration. Degenerative changes in the kidney epithelium were more advanced. The lungs were congested and edema was greater. In death in four days (seven cases) the liver alteration progressed to actual necrosis with loss of large portions of the central vein area, collapse of the sinusoids and hemorrhage. The few remaining cells contained fat vacuoles and appeared degenerating also. All of the cases at this time showed nephrosis. The hepatic, and renal changes were the outstanding features. The other fairly constant findings were pulmonary congestion and occasional small hemorrhages in many organs.

In death in five to seven days (six cases) there were no additional features. There was seen a diminution in the intensity of the previous changes. Five of the six cases autopsied at this time showed early bronchopneumonia. This is an important factor for those treating burns to remember. The adrenals were still congested and there was one duodenal ulcer in this group.

In death in eight to ten days (four cases) very interesting changes were found. There was evidence that healing was taking place in the liver. The liver cell débris was gone. There was still some fat in the

periportal region, but in the center of the lobule regenerating liver cells were present. There was some hemosiderin deposition in the Kupffer's cells. The kidneys, however, showed more advanced changes than seen before. The convoluted tubules were dilated, epithelium was degenerating, and granular casts appeared in them and in the collecting tubules.

In death in eleven days to twelve weeks (eleven cases) the picture is not so clear. Infection with septicemia had been added in eight out of eleven. The organisms were Staphylococcus aureus in two, haemolytic streptococcus in three, and Bacillus pyocaneus in three. The kidney changes became insignificant.

While the facts as given are in general true, it is only fair to state that there is much variation in the individual picture. Thus, a two-year old child dying of a 20 per cent scald in four days showed extensive liver necrosis, while a five-year old with a 60 per cent fire burn, dying after the same interval, showed only moderate and early central necrosis.

The question of exsanguination transfusion will be briefly discussed. The experience of our hospital in this regard was never published. It was begun in 1920 by Dr. Bruce Robertson, who had a particular interest in burns, and discontinued in 1930. There were eighty-five transfusions performed during this time for burns and still more for other conditions. It consisted in the withdrawal of 50 cc. of blood per pound of body weight and the replacement with a slightly greater amount from a number of donors. The withdrawal was started first, and after an interval the two acts proceeded together. Many cases appeared to be improved following the operation. It was because of this that the procedure was continued for ten years. As much as 1,000 cc. of blood were given to a one-year old child which was later repeated. The operation was difficult and lengthy and was attended with a certain mortality rate directly attributable to it. Thus it was finally discontinued.

Certain other facts concerning the cases are worth mentioning. Many years ago there was a 10 per cent incidence of surgical scarlet fever in the patients with burns. Since the introduction of a routine dose of scarlet fever antitoxin, there have been two cases only in considerably over 1,000 burns. One of these received the prophylactic dose. The other was an infant to whom it was not given as it was planned to discontinue its use after the inception of the sulfonamides.

Five cases only developed visible jaundice. Two of these recovered. There were only two cases of gross hematuria. Many cases

vomited bright red blood. This did not mean necessarily a deep duodenal ulcer. Postmortem examination usually showed a superficial erosion of the lower part of the esophagus, stomach or duodenum. About one in ten admitted cases since 1928 required skin replacement. While grafting has been performed in less than one month from the time of the accident, following tanning, this is the exception, and six weeks is nearer an average.

FIG. 1. S. G., age six years; suffered 60 per cent burns. The patient died in fifteen days from sepsis.

Three cases of gangrene of the fingers have been seen. None of these were treated by tannic acid. It is thought that the very great reduction in peripheral circulation together with the trauma is probably the main cause of this complication and not the treatment. Recently in the literature the firm tannic acid eschar has been condemned in this regard.

While it is obvious from the statistics that all patients with burns of large surface area have died, attention is drawn to the fact that many of the patients with the largest fire burns, which are presumably deeper on an average than scalds, lived for considerable lengths of time. One such example, of which there were many, is shown in Figure 1.

Concerning the value of cortin, we are unable to make any definite statement. It has been used since 1939 in fourteen cases. It has been reserved because of expense for those cases in which it was surmised that death might ordinarily be the end result, and for those in which the clinical picture was very serious even though the extent of the burn was not so great. It was started frequently after a number

of days and only when the patient became suddenly seriously ill. In such a case there was always a rapid improvement, which, however, might last for a few hours only. This improvement was judged by clinical observations, there being a return from unconsciousness often, a raised blood pressure, from one which could not be recorded, etc. Cortin* is supposed to be free from pressor or depressor substances. In the cases of burn it has been used in large doses. For example, a three-year old boy with a 25 per cent fire burn was started twelve hours after the accident on 25 cc. intravenously and continued with 5 cc. every two hours for four days. This means a total of 80 cc. in the first twenty-four hours, and 60 cc. a day thereafter. He did not show the expected degree of hemoconcentration, nor did he show the usual clinical signs in great severity. However, as stated previously, while the impression is that cortin is of value, no definite inference can yet be drawn from our cases.

In conclusion, interesting features from over 1,800 acute burns of which approximately 1,000 were under my care have been discussed. In the last three decades there has been a progressive rapid fall in mortality rates, both when based on numbers of hospital admissions and on area burned. The first great reduction (35.2 to 16.1) was coincident with the use of tannic acid. Following this there was a gradual reduction until the final period when there was again a sudden rapid reduction (11.8 to 2.9 per cent). This was coincident with more intensive early general treatment, but other factors may also have played a part. It is thus emphasized that intensive early general therapy must not be neglected in large burns; also while there is as yet no ideal local application for all burns, the changing of a large wet wound, often painful, into a dry comfortable surface (with such a substance as tannic acid) remains an excellent form of therapy. Infection has been a negligible factor in mortality in recent years and a minor cause for morbidity. It is not believed that the number of cases proceeding to grafting after coagulation therapy is greater than after other types of treatment.

Thanks are due to Dr. R. I. Harris for his records until 1928, and also to the Department of Pathology for their meticulous reports. It is to be hoped that the latter will make on their own account a more comprehensive publication of their material.

* The cortin used is manufactured by the Connaught Laboratories, University of Toronto, and dispensed as a sterile aqueous solution, biologically standardized to contain 30 dog-units per cc.

DISCUSSION

J. HUBER WAGNER (Pittsburgh, Pa.): The thought came to me, as I saw these wonderful statistics presented of this marked drop in death rate due either to shock, shock toxemia, or what you will within five days or forty-eight hours, that we cannot attribute this all to the method of treatment but to the fact that burns are being recognized as major surgery and the patients are now being sent to hospitals, whereas in previous years many of these were treated and died at home. The minor burns that were once treated at home are now being treated in a hospital; therefore we know that as the number goes up, the classification is more widespread and it gives a chance for these statistics to be brought down. I am not belittling the paper in any way or the method of treatment, but the fact remains that that is a possibility.

The fact still remains that burns are major surgery and if treated as major surgery such as we know with these modern aids, transfusions, plasma and proper local treatment, our results will be better.

I should like to have heard the statistics of the number of days in the hospital, but that was not available. But we do know that in many of our cases of burns, first and second classification, the stay in the hospital is very limited, whereas with a third or a little beyond a third, a fourth, if you will, if the burned area does not extend over 40 per cent, with our modern methods we have an excellent chance of saving the child's life, and with later skin grafting, of bringing him around to a normal, healthy individual again.

I am quite in accord with the modern trend as I believe that with the use of tannic acid, there is quite a possibility of tanning some tissue that might, if treated by some milder procedure, still live and be viable; whereas if it is on the border, tannic acid takes away what little live tissue might be left and may possibly cause a little deeper burn in itself.

I think the method of compression treatment that has recently been advocated by Siler is quite in accord with what has been done in our clinic since 1915 and 1916. We still believe that the paraffin wax is an excellent method of treatment. It allays pain immediately, and when properly applied, after the first dressing causes no discomfort whatever.

The eschar method, we know, is certainly of minimum value when compared with tannic acid. The thought that has been brought out by Blair, of compression intelligently applied, with something definite in mind, has, I think, been an excellent contribution. We believe that this modern treatment of vaseline plus the sponge dressings is in a sense what we have been doing with wax for a number of years, and it is my opinion now that it is our tendency, rather than to treat these patients every second day, to carry our wax dressings as long as six or eight days with good effects.

Dr. Farmer's paper is an excellent presentation. I think the work done at Toronto Sick Children's Hospital is as good if not better than that done at

any place I have known. We do not keep statistics. Ours is not a teaching institution, so what we have are mostly impressions rather than the actual facts; but I believe, by and large, that we save as many children as has been shown here, and I know that we are getting many more burns to treat that are less severe than has been the case in the past.

E. Payne Palmer (Phoenix, Ariz.): For the past two years we have been carrying on experimental work in shock, and we believe that the emergency treatment of shock in burns is as important as in fractures. These patients should be treated at the site of the burn. First relieve the shock and then treat the burn. Many of these patients die of shock on the way to the hospital or shortly after they arrive in the hospital. Therefore, we must have a remedy which we can administer at the place of the accident.

Using a number of chemicals and drugs, we have found that we have had only one that has given us satisfactory results. These experiments which we have carried out show that there has been a depression of the muscle tone, a depression of the venous system and a slowing down of the blood current, and the only remedy that we have been able to give that could stimulate muscle tone, lowered venous tone and bring about a return of the venous flow has been coramine. I will not give you the chemical name because it is rather complicated. We found that it is necessary to give this in large doses, both to children and to adults, depending upon the age of the child. The adult that is unconscious should get it intravenously and not intramuscularly, because if you give it intramuscularly or subcutaneously, you will get no effect that will relieve the shock.

The conscious patient should be given 5 cc., and repeated 10 or 5 cc. as necessary in the child, in proportion to its age. Shortly after the administration, the patient takes a deep breath, there is an improvement in the color, the patient who is unconscious becomes restless, there is an improvement in muscle tone, in venous tone and in the return circulation.

You can administer this with no fear of toxicity, because if toxicity occurs, you are getting the physiologic effect that you want to obtain. You want to get a muscle tone and push the blood back into circulation. You want to get your venous circulation going. If toxicity occurs, the only thing you are going to get is a slight convulsion which will soon pass away. So to those of you who have not been using this drug, I am not advocating a particular thing except for the benefits which you will derive by the use of this remedy.

Dr. Farmer's presentation has been a wonderful thing. There is one thing that I would like to stress: That tannic acid should not be used on the face and on the hands, that normal saline in your compression dressing would give you the best results. Severe burns should have early skin grafting. Do not wait until a large amount of granulating tissue forms. I would also emphasize early mobility. Have the patient use his extremities early and get the muscle tone back to normal as soon as possible.

ALFRED W. FARMER (closing): I have been asked to detail the factors which I think have played a part in the improvement in the last three decades. Of course, the greatest improvement came immediately in 1925 on the use of tannic acid, and that we believe was due to tannic acid and due to the heightened interest in burns at that time.

Second, the last great drop which came in the mortality rates was since 1938, and I believe that is due to the early treatment we give the patient. As Dr. Palmer has said, we would treat the patient in the first twelve hours or twenty-four hours or forty-hours very intensively, for what may be called secondary shock or toxemia.

Dr. Wagner emphasized that patients with minor burns are now coming into the hospital. That was the reason we made some estimate of the surface area of burn, because that has always been the criticism of any figures which have come out in the literature concerning increase in mortality rates, or decrease in mortality rates, and it is absolutely necessary that one define what he is dealing with in the way of burns as far as surface area, if possible, and, if possible, depth, too; but that we find impossible in a paper of this kind.

Concerning the stay at the hospital, our patients remain in the hospital about two to three weeks, if the burn is not all the way through the skin; and if it is and they need skin grafting, we are able to skin graft them in four to six weeks after they have had tannic acid applied and removed. We have grafted people within three weeks of the time a tannic acid coating has been applied.

Referring to coramine which Dr. Palmer discussed, I do not know anything about it. We administer coramine to those patients who look very sick, but why, I do not know.

TRAUMATIC ARTERIAL ANEURYSMS OF THE PERIPHERAL ARTERIES*

PATHOLOGY, CLINICAL MANIFESTATIONS, DIAGNOSIS AND TREATMENT

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DUE to our participation in the present worldwide conflict, opportunities for observation and the study of all characters and types of wounds will be unlimited. The opportunity to increase our knowledge of vascular pathology will be greatly enhanced by the inevitable traumatic injuries to the peripheral vascular tree. That vascular injuries both systemic and peripheral will occur in a rather high percentage of the wounded is revealed by the statistics of World War I. According to Matas,[1] 20 to 25 per cent of the wounded reaching the first post of surgical relief will present wounds of the vascular system. Therefore, those that survive the primary vascular injury without loss of limb, will have as a sequela to the vascular injury, traumatic arterial and arteriovenous aneurysms. In World War I, Makins[2] compiled 523 traumatic aneurysms from various British hospitals, chiefly from London, while Solomon,[3] in Germany, estimated that fully 1,400 traumatic aneurysms had been operated upon up until 1918.

The incidence of traumatic aneurysms, according to Matas's statistics, ranged from 1 to 2 per cent of the wounded. Therefore, the incidence of traumatic arterial aneurysms is very low, when one considers that the 1 to 2 per cent incidence includes both the arterial and arteriovenous types. If one considers only the pure traumatic pseudo-arterial aneurysm, the incidence will still be lower. There are many factors that are contributory to the low incidence of traumatic aneurysms. Where large vessels are injured, the individual receiving the injury usually succumbs before he can receive first aid treatment. In those cases in which early treatment can be successfully

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given, the bleeding vessels in the majority of cases are immediately ligated. In a rather high percentage of these cases, subsequent infection or ischemic gangrene necessitates amputation. Therefore, in the patients who have early ligation of the injured vessels and in the more unfortunate who lose these injured extremities, aneurysms cannot occur.

Again the anatomical location of the vessel injured plays an important rôle in the development of an aneurysm as a sequela to vascular injury. In the arteries that are more or less fixed in their anatomical position by the surrounding tissue as well as by the numerous vascular branches, they cannot bend or be caused to move out of the path of the projectile, by the pressure produced within the tissue, following the explosive violence of the bullet or shell fragment as it is motivated through the subcutaneous tissue. Whereas when the vessel or vessels are embedded within very loose cellular tissue and the vascular branches are scarce or absent, the vessel can, so to speak, dodge the missile. Therefore, the latter vessels will escape the type of injury necessary for the production of an aneurysm.

The incidence of traumatic aneurysms also will vary according to the vessels involved. Thus Makins states that the highest incidence was in the femoral tract. His statistics relative to the frequency of traumatic aneurysms to the vascular tree were as follows: femoral, 35.2 per cent; popliteal, 17.6 per cent; carotid, 17.5 per cent; axillary, 12.2 per cent; brachial, 9.5 per cent; subclavian 6 per cent and the other vessels less than 1 per cent. The incidences as stated above correspond more or less to the fixation or flexibility of the vessels.

As traumatic injury to the peripheral vessels results in both the arterial and arteriovenous types of aneurysms, only the former will be discussed in this presentation.

Traumatic arterial aneurysms can be classified etiologically as follows: (1) those due to bullet or shell fragments of high explosive shells and hand grenades, (2) splinters of glass and bomb fragments (air raids), (3) those due to stab wounds such as knife, bayonet and sharp, knife-like splinters of glass, and (4) those due to fractures of the long bones that are in close anatomical contact with the main peripheral arteries. The wounds of the soft parts associated with the above types of injury are divisible into punctured wounds and opened and lacerated wounds, the punctured wounds predominating in those cases that develop aneurysms.

The wounds of peripheral arteries secondary to the above types of injury can be classified into four groups (Fig. 1): (1) those in which

a large segment of the artery is destroyed, (2) those in which the artery is completely severed with destruction of the two arterial stumps, (3) those in which there is only partial or incomplete sever-

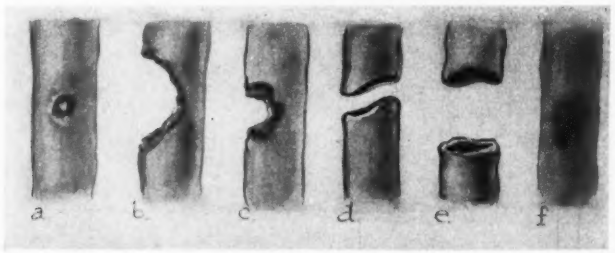

FIG. 1. Drawing showing the more common types of traumatic wounds of the arteries: (a) through-and-through wound; (b and c) lateral wounds with incomplete severance of the vessel wall. This type is dangerous due to the contracting longitudinal fibers which keep the wound open. (d) Complete severance knife wound; (e) destruction of vessel with complete severance, and (f) contusion of the arterial wall.

ance of the artery (lateral wounds), and (4) those in which the arterial wall is contused without primary loss of arterial wall substance.

In group one, in which a large segment of the artery is destroyed, the accompanying wound is usually an extensive lacerated and contused wound. The tissues about the artery as a rule reveal extensive destruction. This extensive destruction of muscles so frequently destroys the collaterals, that the majority of these patients bleed to death before aid can be given. If death does not occur immediately, and the individual reaches a first aid station, the vessels are ligated and the wounds débrided. Traumatic aneurysms seldom if ever develop in this type of injury. However, the percentage of ischemic gangrene necessitating amputations is very high. In group two a similar wound may be inflicted and the sequence of events take place as in group one. However, in group two if the wound of the soft parts is not extensive, bleeding may stop spontaneously by hematoma formation, which may give rise to latent aneurysm formation. Cases of this character, however, should be operated upon immediately, the devitalized tissue removed and the injured vessels resected and ligated. This prophylactic procedure will prevent the subsequent development of pseudo-arterial aneurysms. In group three there may be an open or even an extensive lacerated wound. If the latter is present, a formidable hemorrhage usually occurs and the life of the

individual, unless early treatment is available, is fatal in a large percentage of these cases. The procedure, of course, is similar to the treatment of one and two, early débridement and arterial ligation. However, the patients in this group who are prone to develop arterial aneurysms are those in whom the wound of the soft parts is a punctured wound. The wounds are usually produced by small fragments. They may be from bullets, splinters of shell fragment, high explosive bombs, hand grenades, and splinters of glass in civilian air raids. The hemorrhage is never severe in this type as hematoma formation is rapid and the pressure exerted by the hematoma in the surrounding limiting tissues results in pressure hemostasis. In the majority of these cases conservative treatment is the method of choice.

Conservatism is continued in the management of these cases, with constant investigation of and the evaluation of the efficiency of the blood supply to the tissues distal to the injury. There is one exception to this rule of conservatism and that is in the case in which the hematoma spreads into the tissues without limitation. When this undesirable complication occurs the bleeding into the extremity is excessive. The pressure of this extensive effusion of blood becomes so great that both the main artery and its collaterals as well as the venous system are completely blocked. The obvious results of this complication is gangrene of an extremity. This type of lesion demands immediate operation (provided shock is relieved as should be in all vascular injuries before any operative procedure is instigated) to stop the flow of blood by arterial ligation or suture and to relieve the increasing interstitial pressure which is of paramount importance. In group four, in which the artery is only contused, the incidence of aneurysms will be almost negligible. In the majority of cases of severe contusions of the arteries the intima is ruptured and intraluminal thrombosis invariably occurs very rapidly. The artery then becomes blocked at the site of contusion. Organization and recannulization rapidly follow in sequence, resulting in local strengthening of the vessel. However, where the contusion has been of less violent character, hemorrhage occurs in the wall of the artery. The bleeding seeps in between the muscle bundles and interstitial spaces, resulting in deficient localized nutrition and necrosis, the latter varying in its degree. It is, therefore, conceivable that this destructive process will precipitate a weak area in the arterial wall. Some weeks or months later this deficient area will stretch and balloon out producing a sacciform type of arterial aneurysm, or the wall may

rupture and produce a false aneurysm. This type of aneurysm has a high incidence of secondary rupture, due to the thinness of the damaged arterial wall. When this occurs, a secondary type of pseudo-aneurysm develops which has all the gross and clinical characteristics of the true traumatic type. Therefore, in all cases of wounds of the soft part in which the artery has not been severed but only contused, one should estimate carefully the direction and proximity of the wound to the peripheral vessels. If in one's opinion there has been arterial contusion (usually diagnosed by segmental spasm or thrombosis resulting in diminished or absence of pulse below the site of injury), the case should be under constant observation for secondary arterial rupture and aneurysm formation.

In all cases in which there has been direct severance and destruction of the arterial wall the concomitant vein is invariably injured. This converts both the arterial and venous channels into a common pool. When this occurs, an arteriovenous aneurysm is formed. The type of wound that is productive of arteriovenous aneurysms is in the majority of the cases a punctured wound. If injury to both artery and vein occurs in an open and lacerated wound arteriovenous aneurysms seldom if ever develop. It is the small punctured wound produced either by a bullet, small fragment of bomb, or shell, or splinters of glass that is responsible for the highest incidence of both arterial and arteriovenous aneurysms of the peripheral vessels. The high incidence of arteriovenous aneurysms following traumatic punctured wounds of the peripheral vessels is not within the scope of this discussion. However, attention must be called to their frequent occurrence in order that those called upon to treat vascular injuries in the present conflict will be constantly reminded of their occurrence. They are more dangerous to life than are the arterial type, because of their severe local and systemic pathologic physiology, their operative difficulties and the hazards encountered in their surgical treatment and cure.

The pathology of traumatic arterial aneurysms varies considerably as regards the formation of their limiting walls and their peculiarities. Following the injury of an artery by a missile there occurs an immediate hemorrhage within the tissues which varies from profuse, moderate, to slight with or without external bleeding. The hemorrhage within the tissues may be divided into two types: (1) diffuse and progressive and (2) limited. Type one, as shown in Figures 3 and 5, occurs in areas where muscle planes form large spaces (popliteal) without limiting tissues. This type due to its

increasing pressure, compresses the artery at the site of the injury as well as the main artery distal and proximal to the injury including the collaterals. The incidence of ischemic gangrene in type one is very

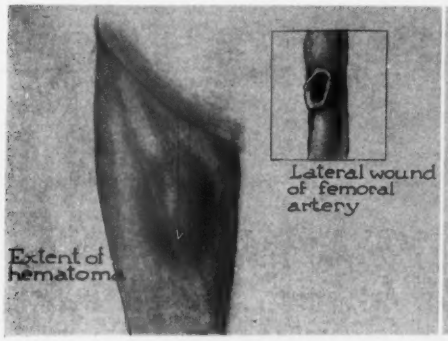

FIG. 2. Lateral wound of femoral artery with localized hematoma formation.

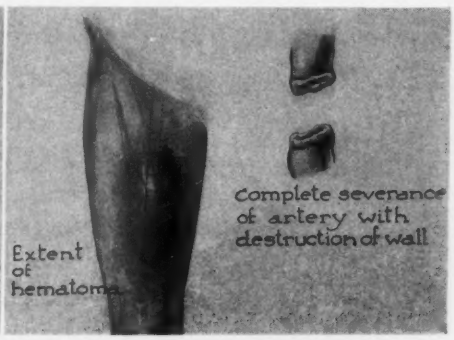

FIG. 3. Complete severance of femoral artery with large diffuse hematoma.

high, resulting not only in loss of the extremity involved but life as well.

Type two is that in which the extravasated blood is confined to a limited perivascular space by the surrounding musculo-aponeurotic planes resulting in a circumscribed hematoma. (Figs. 2 and 4.) This circumscribed hematoma may or may not pulsate synchronously with the heart beat. If pulsation is present, the lesion is then called a pulsating hematoma and is easily diagnosed. If, on the other hand, there is no pulsation, it simply signifies that the pressure of the hematoma is so great that it prevents escape of blood from the arterial injury. It is in the latter type that the aneurysm is delayed in its development for days or weeks following the initial injury.

Hematomas that are formed by vascular injuries may terminate in several ways. When the arterial lesion is small and the surrounding tissues limit the size of the hematoma by compression, the opening may be plugged by a clot. This stops the blood flow; organization follows with healing of the artery and absorption of the hematoma. The above sequence of events, however, seldom occurs either in military or civil practice, therefore, spontaneous recovery is the exception to the rule unless the arterial opening is of minute size. The hematoma may become infected, especially in those cases of punctured wounds, producing a phlegmonous process which varies in degree and extent depending upon the type and virulence of the invading organism. When a perivascular hematoma becomes infected, secondary hemorrhage invariably follows. It is most impor-

tant that the surgeon should be cognizant of the grave eventualities that may occur on opening these infected suppurating hematomas. Following the relief of tension there may occur a violent and uncon-

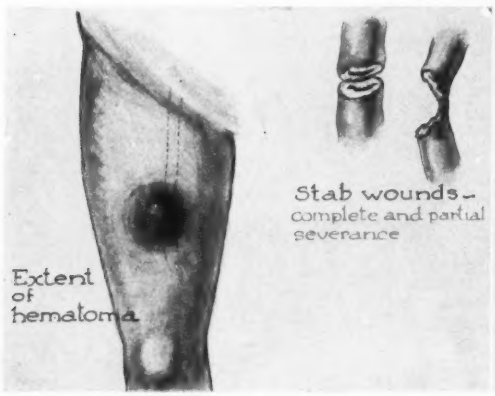

FIG. 4. Incomplete severance of arterial wall of femoral artery demonstrating a small firm localized hematoma.

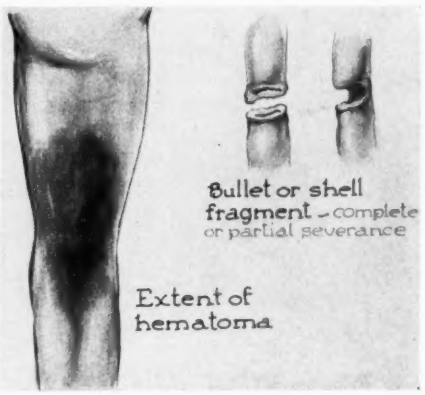

FIG. 5. Wound of popliteal artery with extensive desiccating hematoma formation. This type of diffuse hematoma frequently compresses both the main artery and the collaterals.

trollable hemorrhage with sudden death. An infected hematoma associated with vascular injury should never be opened unless adequate precautions for the proximal control of the blood supply have been provided for in a most secure and efficient manner. This proximal control of the blood flow is of inestimable value in injuries of the large vessels at the root of the extremities and the base of the neck.

When the hematoma gradually spreads in the fascial and muscle spaces (popliteal and along the sciatic nerve to the gluteal region), the pressure produced by the blood clot increases rapidly in the tissues and compresses and obliterates the collaterals as well as the main vessel. When this takes place there occurs a rapid ischemia of the tissues and gangrene with loss of an extremity ensues. In other instances, the patient actually bleeds to death within his own tissues. There probably occurs a train of events that are produced by a loss of fluids plus total blood elements (shock resulting from leg trauma) that result in shock, which is progressive and irreversible, resulting in death. The author has seen two patients with fracture of the femur with tearing of the femoral artery who bled to death; the bleeding was entirely within the tissues of the thigh. There were no signs of external bleeding in either case as the fractures were not of

the compound type. In these cases of spreading hematoma it is absolutely imperative that immediate surgical exploration be undertaken. Delay in these cases always proves costly in that, not only is loss of limb to be expected, but life itself.

The small compressed and hard hematoma limited in its extent by aponeurotic structures, may cause sufficient pressure on the main arterial stem that it acts almost in the same way as an embolus. There occurs an abrupt localized obliteration of the arterial lumen, which inhibits blood flow distal to the obstruction. The collaterals do not have sufficient time for their development, consequently ischemia of varying degrees occurs. The resulting pathological condition ranges from focal muscle necrosis to segmental or massive gangrene. When these small hematomas compress and obliterate the main artery (usually the veins suffer the same pressure and obliteration), immediate surgery is indicated to release the pressure. The hematoma should be evacuated from the tissue and the injured vessel dealt with either by suture or ligation. In war surgery, ligation is probably the procedure of choice, because arteriorrhaphy is too great a time consumer. The results of arteriorrhaphy in the majority of instances are excellent, i.e., restoration of pulse distal to the suture. However, if arteriography is resorted to, one will be impressed by the greater majority of circular sutured vessels that show obstruction at the suture line, the return of the pulse distal to the arteriorrhaphy being due entirely to blood flow through the collateral circulation and not through a restored lumen at the site of injury. Therefore, I would not only suggest but approve and recommend the procedure of ligation instead of suture, in the great majority of cases of injury to the peripheral arteries. *In either ligation or suture, the sympathetics to both the upper and lower extremities, dependent upon the anatomical location of the wound, should be "blocked out" by repeated novocain injection until the circulation has been restored and maintained by the development of the collateral vessels.*[4]

From the foregoing discussion of injuries to the arteries with hematoma formation, one can readily understand that a comparatively large number of these patients will subsequently develop traumatic aneurysms. When an artery is directly injured by a projected missile, three things may occur which have a direct bearing on the type of sac that is formed. When the artery is only partially severed, the hematoma surrounds the site of the injury resulting in a traumatic aneurysm which, in the majority of cases, is of the sacciform type. It is in this type of aneurysm that the communication

from the artery to the sac is a small opening in the arterial wall. Therefore, for practical purposes, almost the entire wall of the artery is present and the aneurysm sac sits (so to speak) upon the wall of the artery. This is the ideal type for restorative endo-aneurysmorrhaphy of Matas. Incidentally, in this type the main blood supply to the distal parts is through the main artery. Therefore, the collaterals are infrequently developed in the cases of sacciform aneurysm.

When the artery is completely penetrated by a missile, which traverses one wall and exits through the other (a rather rare injury) the artery has two openings with an intervening arterial wall. This type of injury also gives rise to a sacciform aneurysm, the sac being bilocular. This type, although sacciform, is more prone to have some development of the collaterals but is not remediable by a restorative type of endo-aneurysmorrhaphy. The obliterative type of endo-aneurysmorrhaphy is, therefore, the procedure of choice. When the artery is completely severed by a missile or knife, a large intervening and surrounding hematoma is quickly formed. The majority of these cases lead to the formation of the fusiform type of traumatic arterial aneurysm. There is a great variety of sac formation in the above type, the character and extent of the sac corresponding to the limiting muscles and fascial planes. In some instances the sac may be oblong or spindle-shape on one side with a pseudopod projecting on the opposite side. In other words, the anterior wall of the aneurysm sac may be spindle or fusiform in shape, whereas on the posterior aspect the posterior wall may be sacciform. This is frequently true in the lower third of the femoral arteries near abductor foramen and also in the popliteal artery. In the popliteal artery the anterior wall of the sac is limited by the posterior aspect of the osseous structures forming the knee, whereas the posterior wall of the sac is limited only by the skin and superficial fascia. Therefore, in aneurysms of the popliteal artery the majority have the gross and clinical appearance of the sacciform type. Not infrequently in aneurysms of the popliteal artery the superior pole rotates posteriorly and downward and the inferior pole rotates anteriorly and upward. It is in the above type that the surgeon if not familiar with the pole rotation will invariably injure the proximal artery in opening the sac as it courses downward on the posterior aspect of the aneurysm.

The formation and development of the sac of traumatic aneurysms is most interesting both to the surgeon and to the pathologist. The artery having been partially, subtotally, or completely disrupted, is immediately surrounded by profuse bleeding and hema-

toma formation which disrupts the surrounding periarterial tissues. The escaped blood rapidly coagulates forming a mass which by its compression on the surrounding tissues and vessels acts as a hemostatic plug. The surrounding tissues are infiltrated with escaped blood and the spread of the blood coagulum is limited by the surrounding musculofascial planes. However, the blood from the injured artery continues to be pumped into and against this coagulated blood clot. The elastic blood clot is then molded and stretched by the systolic force of the escaping blood until the blood clot conforms to the shape of the surrounding muscles and fascia which act as a barrier against further spread. The blood escaping from the injured artery maintains a liquid state in the clot immediately surrounding the artery. Eddies and whirlpools are set up in this liquid center which have a tendency to core out and mold the inner blood clot into a definite cavity. The tissues, surrounding this blood clot with its coagulated periphery and liquid mobile center, proliferate rapidly producing an inflammatory reactive limiting membrane at the periphery of the hematoma. The proliferating tissues rapidly invade the periphery of the blood clot organizing the outer wall of the hematoma, thus forming a definite fibrous wall extending completely around the periphery of the blood clot. This formation takes place rather slowly and is usually accomplished within three to six weeks. While the outer wall is being organized, new layers of clot are laid down at the periphery of the cavity of the hematoma which is continuous with the opening or openings in the artery. Therefore, from without a connective tissue wall is formed while from within layers of new clots are formed, producing a laminated aneurysmal wall. As soon as the cavity of the blood clot is formed and lamination begins to take place, the endothelium from the wound or wounds of the artery begins to proliferate very rapidly along the inner surface of the cavity. This rapid proliferation and extension of the blood vessel endothelium continues until the entire cavity is waxed with shining endothelium. There is now a fibrous connective tissue wall (a true living membrane) reinforced with laminated blood clot which surrounds the endothelialized cavity connecting directly with the artery. A true pseudoarterial aneurysm is thus formed. The entire mass now reveals an expansile pulsation, which varies greatly in character and degree and is synchronous with the cardiac impulse. The traumatic arterial aneurysm differs from the true type in that the walls of the latter contain the elements of the arterial wall; whereas in the former the walls are composed of laminated blood

clot, encased in a fibrous capsule derived from the periarterial adventitious tissue, which contains only the endothelial structures from the injured artery. Therefore, traumatic arterial aneurysms are called false or pseudoarterial aneurysms and the spontaneous ones are called true arterial aneurysms. Their clinical manifestations, behavior and treatment are similar.

The clinical manifestations of traumatic aneurysms of the peripheral arteries can be classified into two groups: (1) immediate and (2) late; both of which can be further classified into local and regional. The immediate symptoms are those of trauma of the soft parts plus hematoma formation in the line of a peripheral artery. The hematoma formation varies greatly in different cases as shown in Figures 2, 3, 4 and 5. The hematoma produces a rather tense swelling at the anatomical site of the injury, i.e., thigh, popliteal space, calf, neck, supraclavicular space, axilla, arms and forearms. There is no pulsation of the hematoma at first, due to the interstitial pressure which is transmitted to the hematoma. The peripheral pulses may be absent, distal to the hematoma. This phenomenon previously was interpreted as complete block of the main vessel, by the pressure of the hematoma, and secondly, by thrombotic occlusion of the arterial lumen at the site of the injury. To the above must be added another explanation, which has seldom been mentioned in the recent or past literature and that is segmental vasospasm secondary to the arterial injury. (Gage,[5] Ochsner and DeBakey,[6] Leriche.[7]) The trauma to the artery precipitates vasospasm which gradually spreads over the vascular tree of the extremity involved. (Haimovici.[8]) If this condition is not recognized immediately and specific therapy instituted, grave and irremediable consequences ensue. The local signs as described above are the ones observed by inspection and palpation which in themselves are most suggestive of vascular injury. On auscultation one may or may not hear a soft blowing systolic murmur. The presence or absence of a murmur is dependent upon the fluidity of the hematoma and its exerted pressure upon the involved vessel. Sometimes the murmur does not manifest itself for hours or even days following the vascular injury. However, when once established, it is pathognomonic of arterial aneurysm. It is considered the one infallible sign of arterial aneurysm when present. Therefore, when one suspects an injury to a peripheral artery, this sign should be frequently sought for; and if the main peripheral vessel is partially or wholly severed, the observer will be rewarded by this distinct and characteristic audible auscultatory sound.

The bruit at first is soft and blowing in character but as the aneurysm sac gradually takes form, the limiting membrane contracting, the blood clot becoming desiccated and laminated, the bruit gradually changes until a loud whistling or harsh systolic sound is heard. This systolic murmur or bruit varies considerably depending upon the size of the vessel, the size of the aneurysm sac, its elasticity and above all the size and type of opening or openings in the associated artery. However, with the changes in sound and pitch of the bruit one feature is always constant, and that is the murmur or bruit regardless of type is constantly and specifically systolic in time. This is always pathognomonic of the pure arterial aneurysm. This type of arterial aneurysmal murmur should never be confused with the continuous hum or roar with systolic exacerbations which is so characteristic of arteriovenous aneurysms. The variations in murmurs of all forms of aneurysms is one of the most interesting chapters in vascular surgery.

The propagation of the murmur in arterial aneurysms very seldom extends any considerable distance. The murmur remains localized, being transmitted distally only for a very short distance. This is also in contradistinction to the murmur of arteriovenous aneurysms which is transmitted over a wide area both peripherally and centrally. There is, however, a cardiac murmur in arterial injuries that was described by Makins[2] during the last war. This systolic cardiac murmur was heard in thirty-seven out of a total of 180 cases of arterial injuries. Of these thirty-seven cases in which the cardiac murmur was present, eighteen were arteriovenous injuries and nineteen were pure arterial. Therefore, in all suspected vascular injuries or in all cases of wounds, the presence of a systolic cardiac murmur is indicative of a peripheral vascular injury. The presence of a vascular injury should, therefore, be diligently sought for, not once, but many times, until one is completely satisfied that an arterial injury is nonexistent.

The late signs are practically the same as those described above with the exception of those demonstrated by palpation. As the tissues proliferate and contract the hematoma becomes progressively smaller until a minimum is reached. This is due to the buffeting of the fibrous wall by intrasaccular lamination and desiccation of the blood clot which prevents further shrinkage. When this stage is reached, usually within six weeks to three months after the injury, a well developed and defined traumatic arterial aneurysm is formed. One now finds a definite expansile tumor in the line of the artery

involved. The expansile pulsation is systolic in time and is synchronous with heart beat. The tumor mass is compressible to a certain degree, depending upon the amount of the intrasaccular laminated clot. Where the latter is thin, the compressibility is great. When the intrasaccular clot is very thick the compressibility is almost negligible. Therefore, the compressibility and expansile pulsation depend entirely upon the thickness of the intrasaccular laminated clot. This certainly is not properly appreciated, with the result that many cases of arterial aneurysm are not diagnosed.

Another sign that is of great diagnostic importance is the one in which the aneurysm decreases in size and stops pulsating when the proximal artery is obliterated. When the proximal artery that enters the aneurysm is compressed, the sac gradually decreases in size and all pulsations cease. If one auscultates the aneurysm while the proximal artery is compressed, no sounds are heard. The above signs are the most convincing proof of the presence of an arterial aneurysm.

Still another sign of less importance than the above is the diminution of the size of the aneurysm sac on elevation of the extremity in which it is located. As this only applies to aneurysms in the extremities, its usefulness as a diagnostic procedure is limited.

The regional symptoms are few and pertain mainly to the vessels distal to the point of arterial injury. If the arteries pulsate normally distal to the injury, it signifies that the blood supply to the tissues is through the main arterial channels. When the pulsation of the arteries is diminished, it is demonstrative that some of the blood flow is being shunted through the collaterals. If there is no palpable pulsation in the arteries distal to the injury, one is justified in assuming that the blood supply to the tissue is mainly through the collateral vessels. The latter condition is known as Delbet's sign which is of great prognostic importance because it indicates that an adequate collateral circulation has been developed. The successful cure of the majority of arterial aneurysms depends upon an adequate and sustained collateral circulation. Because the main artery must be obliterated, the importance of the above sign is readily understood. Occasionally, other peripheral signs manifest themselves in the form of some of the more common diseases of the peripheral arteries. When an embolus breaks off from the clot in the aneurysm sac, it frequently blocks one of the peripheral arteries. When this occurs, signs and symptoms are produced which simulate Buerger's disease. In some of the cases the sudden blockage of the artery distal to the aneurysm by an ambolus from the sac results

in gangrene of the tissues. The extent of the gangrene varies considerably in degree from small areas of necrosis to segmental and massive gangrenous processes. Therefore, one is occasionally confronted with a case of arterial aneurysm with signs and symptoms of Buerger's or Raynaud's disease. The aneurysm in the above type of case frequently goes undiagnosed. All cases presenting signs of peripheral arterial disease of the lower extremities should be investigated for aneurysms of the peripheral arteries. Recently the author saw a case which had been diagnosed as bilateral Buerger's disease of the lower extremities. There was gangrene of the fourth and fifth toes of the right foot with reddish cyanosis of both feet. On careful examination it was discovered that the patient had bilateral popliteal arterial aneurysms. The gangrene was due to embolic occlusion of the anterior tibial artery. The embolus had its origin from the thrombus within the aneurysm sac. The above case is cited to emphasize the ease with which serious mistakes in the diagnosis of aneurysm can be made.

Before any operative procedure can be applied to the traumatic arterial aneurysms one must first determine the presence or absence of an adequate collateral circulation. This is necessary because a successful operation for the cure of an aneurysm depends primarily on the presence of a collateral blood volume flow, sufficient to maintain normal metabolism to the tissues distal to the aneurysm, because the main artery will be obliterated at that site. If the collateral circulation is not adequately developed, gangrene will occur in from 5 to 15 per cent of the cases. Therefore, the first thing to do is to test the collateral circulation in all aneurysms of the peripheral arteries. This is best accomplished by the Matas method. This method has been repeatedly described by Matas[11] and the author.[4,5] The test is performed as follows: In the extremities a rubber bandage is applied from the toes to just above the aneurysm. This milks all the blood out of that part of the extremity covered by the bandage. The Matas compressor is now applied to the main artery just above the point where the rubber bandage stops. The artery is obliterated by the compressor and the rubber bandage quickly removed. The time factor between the removal of the bandage and the appearance of a florid blush to the cadaveric tissue demonstrates whether the collateral circulation is abundant or deficient. If adequate, the blush of the tissues occurs within three minutes. If deficient, the time element is longer; therefore, the greater the time element the less efficient is the collateral circulation. If the test demonstrates that

the collaterals are not developed, the operation must be postponed until the collaterals can be developed.

There are two methods that can be used to develop the collateral circulation: (1) the mechanical and (2) the physiological. The mechanical consists of daily application of the Matas[11] compressor, increasing the time of compression each day until the collaterals are developed. The physiologic[4,5] consists of removing the sympathetic vasomotor control from the peripheral arterial tree. This is accomplished either by mechanical or chemical section of the regional sympathetic ganglia and their connections. This method is preferable to the others because it can be accomplished in three to five days, whereas the other methods require weeks and sometimes months. The rationale of sympathetic block in the developing of the collateral circulation is based upon experimental data collected from the literature as well as the author's personal experience in peripheral vascular disease, including aneurysms. The fundamental principles involved are as follows: (1) overcome vasomotor spasm, (2) produce vasodilatation, (3) increase blood volume flow through the capillaries, (4) increase blood volume flow through the main arterial channel distal to the ligation, (5) increase the size of the vasa vasorum, (6) increase lymph flow, and (7) prevent ischemic gangrene. All of the above conditions occur within three to five days following sympathetic block with novocain and alcohol. The collateral circulation will be adequate and be sustained postoperatively which will prevent the occurrence of ischemic gangrene. Therefore, when the collaterals are developed and efficient, the aneurysm can be safely operated upon.

In the carotid group, the compressor is applied to the carotid to test the efficiency of the collateral circulation to the corresponding cerebral hemisphere. The carotids cannot be safely ligated until the patient can stand obliteration of the carotid for at least one-half hour. To develop the collateral circulation for the carotid, a combination of both the mechanical and physiologic methods is used. The stellate ganglion can be injected from the anterior or posterior approach. The physiologic method is also advocated for aneurysms of the subclavian, axillary and brachial arteries.

The surgical treatment of all arterial aneurysms of the peripheral arteries is based upon the sound principles promulgated by Matas[9] in 1903. He described at that time a new type of operation which revolutionized the surgical treatment of aneurysms of the peripheral arteries. The operation consisted of intrasaccular closure of the

arterial mouths feeding the sac, with obliteration of the aneurysm sac. The operation was described as an endo-aneurysmorrhaphy. He divided the operations into three types: (1) the restorative, (2) reconstructive, and (3) the obliterative endo-aneurysmorrhaphy.

The restorative type is applicable only to the true sacciform aneurysm where over two-thirds of the vessel wall is intact, the opening in the artery being produced by a small lateral wound of the vessel. In this type the sac is opened and the small opening closed by interrupted sutures of silk or cotton. The excess sac is then removed and the remaining portion used to reinforce the arterial wall at the site of the sutured artery.

The reconstructive type of endo-aneurysmorrhaphy was advocated for fusiform aneurysms. The technic was to construct a new channel out of the walls of the sac. It was soon discovered that this procedure was limited in its scope and was finally abandoned because of the high incidence of postoperative thrombosis in the newly created channel.

The obliterative type has become universally applicable to all types of arterial aneurysms of the peripheral arteries except the pure sacciform type. The procedure is of the greatest simplicity. It consists simply of opening the aneurysm sac and closure of the arterial openings of which there may be several, then obliterating the sac. All suture material should be either cotton or silk, as absorbable sutures will give way with dire consequences. In performing the operation it is not necessary to dissect out the sac or either the proximal or distal arteries; one simply exposes the anterior or posterior wall of the aneurysm. The sac is then incised and the edges retracted widely, thoroughly exposing the interior of the sac. All clots are now removed, the sac cleansed thoroughly, and then inspected for the arterial openings. It must be insisted upon that the entire inner surface of the sac be inspected most carefully because if one fails to do this, one or more small openings may be overlooked. Each opening found is closed with interrupted silk or cotton sutures. When all openings have been closed, the tourniquet is released to see if any of the sutured openings leak. This is most important because if the operation is finished before the removal of the tourniquet, bleeding may occur, even another aneurysm may be formed. If no bleeding from the sac is encountered following removal of the tourniquet, the sac is obliterated and the wound of the soft parts is closed by interrupted sutures of cotton. This operative procedure, as described by Matas, is simple in its execution, does not interfere

with the perisaccular collaterals, obliterates the vessels entering into the formation of the aneurysm and destroys the sac. It is, therefore, the ideal operation for aneurysms of the peripheral arteries.

The operation of Matas is, however, not applicable to all aneurysms of the peripheral arteries if we include the carotids, subclavians, the external iliacs and the common femoral. Aneurysms of the above arteries in the majority of instances are best treated by proximal polar ligation, Anel's operation. Polar ligation of the proximal artery is used mainly for aneurysms of the carotid and subclavian groups. Ligation of the subclavian and innominate artery proximal to the aneurysm offers considerable technical difficulties when approached from above. To overcome these difficulties, Touroff[10] recommends that the subclavian be ligated transpleurally. He reported a case of congenital arteriovenous aneurysm of the left upper extremity in which he ligated the first portion of the left subclavian artery transpleurally. He recommends that in traumatic injuries to the subclavian they be treated by transthoracic, transpleural ligation. The author subscribes to this operation and has successfully ligated the left subclavian, transthoracic-transpleural, for an arteriovenous aneurysm of the first portion of the left subclavian. This same procedure is recommended by the author for aneurysms of the right and left subclavians and the innominate and the low common carotids. However, for aneurysms of the first cervical portion of the common carotids, distal polar ligation has been the operation of choice. It would be preferable to use the approach by Touroff.[10]

In traumatic aneurysms involving the external iliac and common femoral the transabdominal ligation of the external iliac is the procedure of choice.

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DISCUSSION

LOUIS G. HERRMANN (Cincinnati, Ohio): Dr. Gage has told us many of the important problems concerning the injury to major arteries in the extremities. He has talked largely about gunshot wounds and stab wounds, and I think we all now believe that reconstruction, either by the Matas' procedure or by Dr. Gage's, is by far the best method of dealing with such things. However, I would like to add one thing, and that is with regard to the cure of aneurysms which result from disease of the wall, especially the syphilitic aneurysms that develop in the popliteal space. It is extremely difficult, and sometimes even dangerous, to attempt to reconstruct them or obliterate them by the Matas' procedure, so that we as a rule attempt to open the sac with the supply of blood controlled by a tourniquet, and then go outside of the sac and isolate the incoming and outgoing arteries, and ligating them high above and high below the sac rather than try to obliterate them at the sac itself.

We have had some recurrences when we have tried to obliterate them by the Matas' procedure alone.

There is very little else to add to what Dr. Gage has already told us. I think he has shown us very definitely that at least traumatic injuries to blood vessels should be repaired with nonabsorbing materials. I think most of the experimental work now will substantiate the fact that catgut, although it does not in many cases, is more or less a dangerous type of suture to use, and that either silk, cotton bandage, cotton tape or some such nonabsorbent material should be used.

There are many factors that Dr. Gage did not have time to touch upon. That is, there are other methods, of course, of developing collateral circulation, and the test that Dr. Gage has described, namely, Dr. Matas' test, is an extremely valuable one; and we believe very definitely that no patient should have a large peripheral artery ligated unless we are certain that the collateral circulation is capable of carrying on.

A little trick that we frequently use for a day, two or three days, or maybe a week, is to insist that the patient compress that artery with his finger in order to promote a more efficient collateral circulation. In young patients and in healthy patients, such a collateral circulation could be developed without mechanical means. The blocking of the sympathetic

chain is a simple thing, as Dr. Gage has told us, and is a very valuable thing. I would stop, however, with the novocaine and not inject the alcohol, but that is just a matter of personal opinion.

The other means of developing collateral circulation which we use in our own clinic have proved valuable and in the last ten years we have not lost any extremities after ligating large vessels, including large vessels of the popliteal space. It is fairly simple to deal with a large aneurysm of the sac in the popliteal and in the femoral region. You control hemorrhage first by tourniquet and then ligate above and below the sac and then ligate the collaterals under there, or use a group of sutures and obliterate the incoming and outgoing vessels in the sac itself.

EDGAR L. GILCREEST (San Francisco, Calif.): Dr. Gage has given us one of his clear, concise papers which I think we are all indebted to him for, particularly at this time. I believe in this great emergency, when we are going to have literally hundreds of doctors dealing with aneurysm, that the admonition of Halsted probably should be publicized, so that surgeons will bear in mind the common errors that he called our attention to years ago. I shall read seven of them.

"1. Opening the sac or pulsating hematoma without first making a temporary occlusion of all the possible sources of hemorrhage.

"2. The permanent ligation of a great arterial trunk as a precautionary measure in the search for a distal bleeding point.

"3. Ligation of a trunk too far from the aneurysm.

"4. Stuffing the wound with gauze to arrest hemorrhage.

"5. Drainage.

"6. The employment of catgut for the ligature, or of silk that is too fine.

"7. Ligation of the artery proximal to an arteriovenous aneurysm or fistula."

In 1929, in a paper on aneurysm, I closed that paper with this remark: "Surgeons attempting to intervene in aneurysms or arteriovenous fistula, should be much more concerned with hemostasis and meticulous technic than with the duration of the operation. These operations are at best always tedious and time-robbing and demand the best of the surgeon's skill. Without producing hemorrhage, these patients stand operations of six or seven hours' duration without severe shock."

And then I quoted Halsted again when he said, "An operator searching for a bleeding point in a pool of blood, and particularly so when embarrassed in his movements by the adherent walls of an aneurysm within which he is working so disadvantageously, presents a distressing spectacle." He said, "I would rather devote an additional hour or more to an operation than be caught for a few moments in such a predicament."

I should like to present a case of an arteriovenous fistula. This patient was walking down the street in Mexico City and was shot by a bandit, the bullet entering the right clavicle. That happened in December, 1920. The

arm did not swell much until early in 1927. During that time, no one had ever told him to elevate his arm, even at night. After he had elevated the arm for six weeks and gotten the edema out of the arm he was operated upon. At the operation I removed one of the clavicles, which is so important. It was Thompson of Galveston who perhaps contributed much on this subject, and he was one of those who has called our attention to the great importance of ligation of the first part of the subclavicle. I think that was due to the fact that in those days, they did not remove the clavicle, which makes this operation much easier.

In this operation I ligated first the innominate vein, the subclavian and jugulars. The five ligations done at that time were: the subclavian artery, the subclavian vein in two parts, the external and internal jugular vein and innominate vein.

During that operation, after we made those ligations, the thrill became imperceptible and the bruit became inaudible, and I went out among the adhesions and ligated what we thought was another portion of the subclavian, and we believed we had better stop at that time.

Nine months later he returned because his arm had begun to swell a few weeks before. We had expected that might occur and realized that we must go then way out and do further ligations. The various ligations made in the first operation and in the second operation numbered eleven in all. Dr. Emile Holman showed the necessity of ligating the innominate vein and preventing gangrene in the extremity.

COMMANDER RICHEY L. WAUGH (Boston, Mass.): I want to add just a few more words about the fistula. We have found in treating the traumatic wounds of the upper extremity quite a few of the arteriovenous fistulas. Even during the past three months, we have had two cases, and I would just like to take a minute to refer to those two cases. Both were due to missiles, one a chip fragment occurring to a young man who was working at the Watertown Arsenal, and the other followed a gunshot wound, rather late after the injury.

In the case of the young man who had a chip fragment enter the forearm, within one week he perceived a warmth and a thrill in the forearm. We operated upon this patient and found an arteriovenous fistula between the ulnar artery and the deep ulnar vein. The treatment was, of course, a quadruple ligation.

In the other case, the gunshot wound in the arm occurred ten years previously. It was followed about four years ago with a severe contusion in which there was ecchymosis and soreness for several days, and I believe only a year ago was the patient aware that there was something wrong with the extremity. He noticed, whenever he put his face on his arm, that it felt much warmer. We operated upon this patient.

First, may I say that in both of these cases, with digital compression over the site that would stop the fistula, there was a slowing of the pulse from ten to fifteen. That is known, I believe, as a Branham's sign.

We operated upon the patient and found a fistula between the profunda artery and the vein. This patient also was treated by quadruple ligation. He is now in our hospital convalescing.

JOSEPH E. J. KING (New York, N. Y.): Since Commander Waugh spoke of the arteriovenous lesion, I should like to make a brief remark on the handling of those lesions as proposed by Dr. Matas. We do know that in the last war we had several cases, and I refer especially to the femoral region above the profunda. Those legs had lesions and instead of doing what Dr. Matas later told us to do, we did quadruple ligation. Fortunately, all the legs were saved, but it is so much better to ligate the vein, open it up, sew the opening in the artery, preserve that section of the vein and sew it over to a patch instead of a quadruple ligation. We did not know that during the last war, and I should like to call this to your attention because you may have to do it.

MIMS GAGE (closing): I am sorry I did not have time enough to go into the pathology and the clinical manifestations in the syphilitic type of aneurysms in speaking of the traumatic aneurysms, but to do that would be too time-consuming.

As regards catgut, Dr. Matas advocated many years ago that we not use it because he had had many of those sutures break down.

As far as development of intravenous occlusion in the subclavicle is concerned, I said in civil practice it takes time. It takes from six weeks to two or three months to develop intravenous occlusion by the Matas compressor or Dr. Herrmann's Pavex machine. The sympathetic block happens to be my child and I have to nourish it along over a long road. It does save time because you can block the aneurysm one day and operate upon it the next.

I have had over ten popliteal aneurysms, all syphilitic but one. In that case I obliterated his arteries secondary to block. I have not had any gangrene in those cases.

Most of us have had syphilitic popliteal aneurysms and have had no trouble suturing them.

I do not know of anything, as Dr. Gilcrest has said, that the surgeon is confronted with that gives him more trouble than an arteriovenous aneurysm at the roots of a limb, including the neck if you want to include that as a limb. You have to control not only the artery and the vein on the proximal and distal side, but all the collaterals as well. Very frequently up in the neck arteriovenous aneurysm defeats you; it really whips you down; you cannot make it. I had one I worked upon for two hours and could not do a thing and had to do a transthoracic ligation of the artery.

The majority of arteriovenous aneurysms have to have quadruple ligation.

The thing I wanted to stress is that during the war we are going to have a large number of injuries to the peripheral arteries and I think if the sur-

geons will recognize them as such and operate upon them as soon as they possibly can, ligate the arteries and then turn them over and block the sympathetics with novocaine, they will save many of these limbs. You can do it without alcohol, but you have to repeat that every day for eight to ten injections until the collaterals are developed. You have no time to carry a Pavex machine or a Matas compressor around with you. If you determine that the artery is injured—which you usually can—it should be ligated and the development of an aneurysm prevented. When an aneurysm manifests itself within ten days to two weeks after the injury, it should never be touched until three to six months later in order to let all the tissue become organized. That is a law that Dr. Matas stated many years ago; because if you operate too soon when the tissue is friable, your sutures will not hold, with the result that a disaster will occur in a giving-away of the suture line.

As far as the operation is concerned, it is simple, provided you can control the proximal blood supply. Where you cannot, you have to dissect out the entire affair and control the blood supply below and above by using temporary ligatures. Outside of that, it is simple; but arteriovenous aneurysm is an entirely different chapter and would require too much time.

FROSTBITE IN SHIPWRECKED MARINERS*

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DURING the past eighteen months I have been one of a group of St. John's surgeons privileged to share in the surgical care of a large group of shipwrecked mariners, survivors of the Battle of the Atlantic, who have been landed at our port.

Of this group 75 per cent of all those who required hospitalization suffered from lesions of the extremities produced as a result of exposure to cold and immersion in the icy waters of the North Atlantic. Many of them had been adrift in open boats or on rafts for periods of up to ten days and some had been completely immersed in the water before being taken into the boats.

No definite figures on the degree of cold to which these men were subjected can be, under the circumstances, expected to be available. We do, however, have reliable data on both atmospheric and water temperatures in the areas where these men were exposed during the winter and early months of spring. Water temperatures in these regions vary from 28° to 30°F. in the more northerly regions and up to 35°F. in the more southerly. The atmospheric temperature naturally shows wider variations but in the wind an atmospheric temperature of 0°F. is not uncommon. It is thus obvious that these men were exposed to atmospheric temperatures often below freezing point and to water of a temperature just about freezing point.

In civil practice frostbite has until recently excited but little comment. Brahdy,[18,19] however, reported no fewer than 996 cases as occurring during the years 1933 to 1936 amongst outdoor municipal employees of the City of New York. Successive military campaigns have directed considerable attention to this problem. During the Napoleonic Wars Larrey[1] observed numerous cases. The condition was adequately described during the Crimean War[2] and later by Page[8] during the Balkan Wars. There was some confusion at times as to the etiological factor[8] but in every case they were ultimately recorded as frostbite. During the last Great War large numbers of

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cases occurred and the name "trench foot"[9] was applied to some of these cases.

Other terms have also been applied. Liggett[20] used the term "dermatitis congelationis." Recently, Webster, Woolhouse and Johnston[27] and Greene[29] have applied the term "immersion foot" to describe lesions present in a group of cases similar to our own. I quote from the excellent study of Smith, Ritchie and Dawson[10] upon the pathology of "trench foot": "The applicability of the term frostbite to the condition to be inquired into has been questioned. After consideration we have decided to continue the use of the word as it accentuates the essential causal factor concerned, namely, cold, and, at the same time, is sufficiently comprehensive to include the various local effects which cold may produce."

We turn now to our own cases. It was soon apparent that these men suffered from lesions of varying degrees of severity and that they could be divided broadly into three groups depending upon the type of lesion presenting. The lower extremities were in all cases affected. In a few cases the upper extremities were affected but never independently of the lower. The lesions were not symmetrical in all cases, that is to say, there were instances in which one leg presented what we shall describe as first degree frostbite, and the other what we shall describe as second degree frostbite; and there were still others with second degree frostbite on one side and with third degree frostbite on the other.

TABLE 1

Survivors Requiring Hospitalization	No. of Cases of Frostbite	Percentage with Frostbite
94	71	75

Nearly all patients, irrespective of the group to which they belonged, gave a similar history. They had noticed no pain until they had been rescued. They had been able to move about, so far as movement was possible, in the life boats, but when rescued or shortly after had lost the use of their legs and experienced pain of varying degrees of severity. Some of them had worn ordinary footwear, some only socks, but the presence or absence of footwear did not appear to have any influence upon the occurrence or severity of the lesions. The history of the onset is similar in all respects to that described by Page[8] in the cases seen by him in the Balkan Wars.

Group I. (*55 Cases.*) Some of the cases in group one were able to walk upon admission. They complained of pain in the affected extremities. The feet and legs were swollen, usually to but not above the junction of the middle and lower third of the leg. There was some erythema but otherwise no generalized color changes were noted. All movements were present. The extremities were warm or hot to the touch. Some areas of anesthesia were noted in the feet but these disappeared after admission. We did not study the distribution and extent of these areas.* Circulation was apparently unaffected and palpable pulsation was present in the dorsalis pedis and posterior tibial vessels. There was in many a slight temperature upon admission of 99 to 100°C. which usually subsided within a few days.

TABLE II

No. of Cases of Frostbite	First Degree	Second Degree	Third Degree
71	55 77%	7 10%	9 13%

TABLE III

No. of Cases	Lower Extremities Affected	Upper Extremities Affected
71	71 %—100	8 %—11

In many of the cases small areas of dusky red mottling were present and bleb formation was noted in many of these areas. The blebs commonly contained dark bloody fluid, in some, straw colored fluid. These areas of mottling and bleb formation occurred most frequently on pressure points; thus the metatarsophalangeal joint of the great toe and of the small toe, the malleoli and heel, and the dorsum of the toes were the parts most frequently affected in this manner. This, I think, is of considerable importance when we come to consider treatment.

Group II. (*7 Cases.*) The cases in this group showed a condition similar to the Group I cases but in addition more serious lesions were present. One or more toes was dusky red in color or black; movement in the toe or toes was absent; it was cold to the touch and sensation

* These areas of anesthesia might possibly be due to inflammatory changes in the end organs.

was absent. In shoor, areas of dry gangrene were present affecting chiefly the toes, but in some the more distal areas of the foot were affected.

Group III. (9 *Cases.*) The cases which fall under this group presented a picture similar at first to the cases in Group I and Group II. The lesions described as proper to Group I and Group II were always present but in addition there were important differences. Usually larger blebs were present and these seemed less superficial and occurred higher up in the affected extremity. One important difference was obvious: From the time they came under observation they appeared toxic and a high fever was present from the onset. Temperatures of 103 to 104°C. were the rule and we soon came to realize that a high fever, in the absence of intercurrent respiratory infection, was significant of grave tissue damage. When I reviewed the cases this impression was confirmed. Every patient with a high fever on admission proved later to belong to Group III.

Not long after admission other signs made their appearance which obviously differentiated these cases from those in the other groups. While cases in Group I and Group II rarely showed new areas of mottling following the institution of treatment, the patients in Group III did; and in spite of rigid aseptic precautions, evidence that infection existed soon became obvious. The parts were hotter to touch and more reddened in appearance. Deep, foul-smelling areas of slough developed; tendons and muscles became, in the course of time, exposed and a foul, putrid, offensive odor developed. Thus, a type of wet gangrene was established in the soft tissues of the extremities. Even in these cases pulsation was present in the large vessels and secondary hemorrhage, though never of serious degree, often was present.

I am convinced that this infection was not introduced into the tissues but was present from the onset as evidenced by the fever often present before any break in the surface tissues occurred. This impression is dependent in part upon and is confirmed by the pathological findings of Smith, Ritchie and Dawson.[10] Further it was noted that muscles and tendons sloughed and became necrotic and exposed but there was notable absence of abscess formation in the tissue spaces.

The upper extremities did not in all cases escape involvement but in no case were the lesions observed in the upper extremities more serious than the type of lesion classified as Group I in the lower extremities.

I should like at this point to draw attention again to several facts. The least serious cases showed in the extremities swelling only with or without bleb formation. These lesions were also present in more serious cases. In the more serious cases in Group II, there was in addition mummification of the most distal areas of the extremity, that is to say, in the areas where the extremity had the least mass, and in the most serious cases, in addition to the Group II lesions, there were present lesions affecting the deeper and thicker tissue masses more proximally in the extremity. Thus it seems to me that we can visualize the sequence of events as follows: The first response was swelling in affected parts. Where there was continuation of exposure beyond this stage the most distal parts, where the mass surface ratio is least, where heat loss to mass ratio can therefore be greatest and where the effects of cold can penetrate to a sufficient depth to produce a maximum reaction, the tissue response was sufficiently great to interfere completely wih tissue nutrition and dry gangrene resulted. Further exposure resulted in damage to structures higher up, chiefly tendons and muscles, of a degree sufficient to bring about necrosis of tissue; but because the thickness or mass of the tissues provided a degree of protection to the cold to which they were subjected, this process was necessarily not so complete as in the case of the more distal parts. Therefore, the death or gangrene of the tissues was slower in onset and at first less complete and so produced what we are pleased to call wet gangrene.

It is my belief that this wet gangrene is not, as some suppose, primarily an infection in the tissues but necrosis due to a physical agent, namely, cold, in which infecting organisms played a saphrophytic rôle. I must again refer to the work of Smith, Ritchie and Dawson[10] to explain the presence of organisms in the tissues. I should like to stress here again the fact that where Group II lesions existed that were present upon admission and where Group III lesions occurred they, too, were present upon admission as evidenced first of all by a high fever. In other words tissue responses and tissue damage were dependent upon exposure, not upon subsequent events. This observation is not a new one and was first noted by Page.[8]

I believe that it would be of inestimable value both to the statistical study of these cases and to a proper understanding of the prognosis in the individual case if we were to classify them somewhat as we classify damage caused by heat, according to the degree of tissue response and damage.

Thus I would suggest that where exposure to cold is the etiological factor we retain the term frostbite. Where the lesions resulting correspond to Group I in our cases we should classify them as frostbite, first degree; where Group II lesions are present we should classify them as frostbite, second degree, and where Group III lesions are present, frostbite, third degree. We would thus be able to say that in our cases we saw frostbite of first, second and third degree in the lower extremities, and of first degree only in the upper extremities.

That this classification, at least as regards first and second degree frostbite is applicable to ordinary cases seen in civil practice, is confirmed by study of the types of cases described by Brahdy.[18,19] In civil practice we have seen one case of frostbite where dry gangrene extended above the ankle joint and with wet gangrene above this area in the muscles. This lesion was produced by prolonged exposure to a more intense degree of cold. There might be some justification for classing this as fourth degree frostbite. As applied to prognosis there is little essential difference from our third degree cases. Loss of limb results.

A pathological study of tissues removed from patients suffering from frostbite is necessarily of limited value since it offers only a picture of the end results. There are, fortunately, several experimental studies of great value. Richspler[3] studied the effects of freezing on animal tissues. He found that the pathological changes which resulted from freezing were entirely inflammatory in nature and that there were not specific lesions of the vessels or nerves. Smith, Ritchie and Dawson[10] studied the effects of exposure to cold and wetness upon the shaven extremities of rabbits. They tried insofar as possible to reproduce the conditions under which "trench foot" occurred and their findings are obviously applicable to the cases in our series. Their findings were strikingly similar to those of Richspler. The degree of wetness or cold to which the tissues were subjected made no essential difference to the type of tissue response except quantitatively. Grossly, the tissues responded by edema. Sections taken shortly after its development showed enlargement of the endothelial cells in the intima of smaller vessels and infiltration of the perivascular tissues with inflammatory cells. Connective tissue cells were swollen and there was plentiful deposition of fibrin in the tissue spaces, molt marked beneath the skin papillae. Concomitant changes of an inflammatory nature were observed in the regional lymph-

nodes. Organisms, staphylococci and streptococci were observed in the subcutaneous tissues before any break in the integument occurred. Application of heat to or constriction of the damaged tissues appeared to be harmful and resulted in extravasation of blood. Sections* of tissues removed from patients in our series differed in no important respect from those described by Brahdy.[18]

Bearing in mind the pathology of frostbite, certain general observations on the early treatment of the condition may be made:

It is obvious from the experimental studies described above that trauma to the tissues must be avoided. Rubbing, with or without snow, is certainly harmful. Heat, in any form, must be avoided. This was first noted by Larrey[1] and Davys.[5] In recent articles Greene[24] and Clouston[23] have both stressed these points. There is a tendency by some, to use the Pavex treatment early in the disease. Greene[24] advises against this. On pathological grounds there are serious objections to its use since there is certainly a risk of additional trauma to the tissues. Miscall,[21] in 1937, suggested that paravertebral sympathetic block might be considered without, however, recommending it. Here again, I can see no possible advantage and upon pathological grounds there are, I think, serious objections. Certainly trauma and infection must be rigidly guarded against. In first degree frostbite, rest, elevation and strict attention to asepsis resulted in a rapid amelioration of the condition in our hands. As soon as the cases came under observation this treatment was instituted and in no patient did we observe any progression of the disease while under treatment. Swelling subsided in due course; the pain gradually disappeared and in the majority there was complete or nearly complete restoration to integrity.

There were, however, some in whom a considerable disability persisted. This resulted from persisting thickening and stiffness of the toes, feet, and in one case, of the hands. It is obvious from the pathological data available that this is due to fibrous tissue formation. Since the formation of fibrous tissue is dependent upon tissue reaction it is equally obvious, I think, that anything which can minimize tissue reaction may be of considerable value.†[13,15] Webster,

* I am indebted to Dr. J. E. Josephson, Pathologist, Public Health Laboratories, St. John's, for his invaluable assistance in the study of these tissues. I am also indebted to Dr. W. Roberts for his kindly encouragement in preparation of the paper.

† Thus Lake suggested the use of vasoconstrictor drugs to reduce the head of pressure on the capillaries and Lewis and Love suggested obstructing the circulation in an effort to achieve the same results.

Woolhouse and Johnston[27] have recently employed and described the use of refrigeration of the affected tissues. They have noted immediate improvement following the institution of this treatment as evidenced by improvement following disappearance of the swelling and the reabsorption of blebs.

In the late treatment of these cases, that is to say, when the primary inflammatory reaction has subsided, physiotherapy, undoubtedly has a place and should be employed.

In the treatment of second degree frostbite conservatism should be practised. Rabut,[22] Greene,[24] and Thompson[26] all have observed that the line of demarcation may ultimately prove to be considerably distal to its apparent site. I can recall one case of frostbite, not in the present series, in which the distal phalanx of the finger appeared to be completely mummified. In the course of time the mummified area completely separated and fell off like a glove leaving a complete and normally functioning distal phalanx. In many cases it is obvious, if sufficient time is allowed to elapse, that superficially the area of gangrene is much more extensive than is present in the deeper tissues. In these cases in our series in which amputations were practised I did not observe that any significant time was saved, for in many healing was considerably delayed. It would seem that the ideal treatment would be to allow spontaneous separation of areas of dry gangrene, to trim necrotic bone and permit healing by granulation. This will in most cases serve to preserve the maximum amount of function.

In third degree frostbite amputation must be performed sufficiently high to permit removal of all areas of gangrene. We did not use guillotine amputations but in all cases flaps were cut and in no case was re-amputation necessary because of infection or spread of gangrenous process. There were, of course, some instances in which small areas of slough developed in the skin but these did not prove serious. Since these amputations were life-saving procedures which were performed upon tissues seriously devitalized, we did not think it proper to concern ourselves at the time of amputation with the ultimate problems of prosthesis.*

The problem of prevention of frostbite in shipwrecked mariners is admittedly difficult but it is my opinion that many of these cases can be prevented. I believe that the provision of a form of protection for the lower extremities at least, which would prevent heat loss

* Had dry gangrene been present, prosthesis would, of course, have been the factor in determining both the time and site of amputation.

and at the same time be waterproof, would be a great advancement. Soft sealskin boots might be of considerable value.

Sailors operating in submarine zones are now compelled to wear life-jackets at all times. This is fitted with a signal light. The addition of a waterproof compartment containing protective garments for the extremities should not prove impossible.

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DISCUSSION

Grover C. Penberthy (Detroit, Mich.): Dr. Brownrigg is to be congratulated, I think, on his study of a very unusual group of cases. It is

timely that this subject be presented to this organization, in view of the fact that many may be called into the naval service to handle this type of lesion; and I think we will all agree with Dr. Brownrigg as regards his classification and his conservative treatment which, of course, means supportive treatment from the beginning, early recognition of the lesion that may require more radical surgery, and at the same time conserving as much as possible.

One in civil life has little opportunity to see frostbites such as have been described. However, I am going to present two cases which were treated by Dr. Johnston, a member of this Association, and describe just what was done with these two patients.

The first individual, sixty-five years of age, was admitted to Receiving Hospital about 1 A.M. Three hours prior to admission, he had been drinking, and on his way home met with a snow drift and fell; he could not get up and the policeman eventually found him and brought him to the Receiving Hospital.

Both hands were markedly swollen and there were blisters. Dr. Brownrigg referred to the paravertebral block. I might say that this particular case was treated by the cervicodorsal block after the technic of Ochsner and Gage, and it apparently did something to add to the comfort of the patient as well as improve the general circulation, as Dr. Johnston described it.

The second case was a gentleman who went fishing out on Lake St. Clair. He wore mittens and felt that his hands were warm, but he started to have a peculiar feeling in his hands, so he returned home. Soon afterward he immersed his hands in warm water, which Dr. Brownrigg has warned against, and was later admitted to the Receiving Hospital. This individual also received multiple cervicodorsal injections.

Both cases eventually required amputation of the fingers.

FRASER B. GURD (Montreal, Canada): I am going to get all the credit that I possibly can from this meeting and draw attention to the fact that Dr. Brownrigg was an ex-resident of mine at Montreal General Hospital, and I knew that he had had this rather unique experience with these unfortunate persons.

Another reason may be that perhaps Dr. Marble's reading may have influenced him, because it is a fact, as some of you may have known, that on more than one occasion I have made contributions (or at least so I thought them to be) on the subject of acute bone atrophy, with regard to the late results of these cases, particularly of trench-foot as we saw it in the last war and in a large number of cases following the last war. I feel, myself, that the late phenomena shown by these cases are tied up with the subject of acute bone atrophy.

Of those in the room, Dr. Donald Gordon, Dr. Herrmann and myself have all written on this subject. We are all at the same level with regard to it in that none of us knows anything about it. I shall not attempt to open

the subject of acute bone atrophy or the late results of trench-foot, because that is not what we are discussing.

SURGEON LIEUT. COMMANDER F. M. WOOLHOUSE, R.C.N.V.R. (Boston, Mass.): I should like to report just very briefly on one very small aspect of the treatment of what we still prefer to call immersion foot, because Lieutenant Commander J. C. White, of the United States Navy, who saw our cases and worked with us on them for over a week, has just reported having seen another large group of cases who were picked up in the Gulf Stream off our southern seaboard. These patients show almost identical lesions, and in that case, of course, there is no question of true frostbite.

We had a total of 142 cases who showed lesions similar to those shown by the cases of Dr. Brownrigg, and in view of the explanation of Freeman, that developing gangrene is simply due to the discrepancy between the local tissue demands and the available blood supply, and that the available blood supply could be utilized by decreasing the local tissue demands, it occurred to us that the simplest and most expeditious manner of doing this would be to apply dry refrigeration. We had no other means at our disposal except the use of ice bags, and after wrapping the feet in sterile towels, after careful aseptic and antiseptic preparation of the feet, the feet were elevated and around the sterile towels were placed several ice bags. Our aim was to reduce the temperature of the feet to about 6°c. below the normal skin temperature.

We found pitting edema, great extravasation of blood, and the feet were often bullatious up to three or four inches above the ankles. We found this difficult to do because of the intense reaction to tissue injury. However, the ice bags were changed at intervals from four hours down to twenty minutes, depending upon the demands of the individual case, and we were able shortly to drop the temperature down.

This produced very dramatic early results. In a period of four hours or less, the edema has practically subsided in the mild cases; in a period of one or two days it had subsided in the serious cases and the skin was wrinkled, and these large blebs, many of them bigger than the palm of one's hand that Dr. Brownrigg described, had resorbed without any break in the overlying tissue.

One other dramatic result that was shown was that this was not due to elevation alone. We were running skin temperatures on these people with a dermatherm, and in going from one end of the ward to the other, which took approximately half an hour, a man in the first bed, whose ice bags we had removed, and who had had a bleb on the plantar surface of one foot larger than the palm of one's hand, and which had resorbed under treatment with the ice bags, was left exposed in the elevated position. By the time we got to the far end of the ward, all his edema had returned and the bleb was back to its original size.

I have not seen any of these cases for about two and a half months and cannot report on the late results. Dr. Webster will be able to do that. We

did note some early delayed effects. In the first place, a lot of people asked us why we did not employ methods of sympathectomy or sympathetic block, such as had been advised by the French writers during this present war, but which had given extremely bad results. These limbs are all essentially sympathectomized to begin with. Lieutenant Commander White had found that the vessels in the affected limb were sensitized to adrenalin about the end of the ten-day period, as he has reported previously on limbs sympathectomized for other reasons.

We believe that by this method we can save some tissue. How much, we are not quite sure yet.

Among the other early delayed results are intense pain developing about the eighth to tenth day, and in severe cases no subsiding for three or four days. This is a burning sensation with superimposed severe lancinating pain in the exposed extremities. Other effects were the systemic toxemias that Dr. Brownrigg has reported on. Some of our cases showed a transient hematuria and albuminuria, with enlargement of the liver, and one other patient died with a staphylococcal septicemia following empyema.

Another important point that was noted was that one officer with very severe damage to both feet, which Dr. Brownrigg would have classed as a Group II injury, gave a history suggestive of having had a previous renal syndrome, and he was consequently heparinized. We believe this was responsible for saving considerable of his tissue.

SURGEON LIEUT. COMMANDER D. R. WEBSTER, R.C.N.V.R. (Boston, Mass.): These were extremely fascinating cases to work with, and in the main I think we agree completely with Dr. Brownrigg, except that we cling to our immersion feet term.

I have returned from one of the southern Marine Hospitals where I saw some of these cases that had been adrift in the Gulf Stream where the temperature of the water was between 60 and 70 degrees, and two of these patients already were showing gangrene of the feet.

The individual variation in these cases is peculiar. Some of these men in the same boat, and apparently very similar types of men, will go on to gangrene, and some seem to have nothing at all the matter with them, and it seems difficult to predict the moment they come in which are going to recover and which are not.

I have a few slides* and kodachromes of these cases when they came in, showing the initial stages. I am sorry we have not the follow-ups.

I saw these 142 cases that came in about a week ago. Two patients had lost several toes; one patient had had one foot amputated, and there are two more that should be about ready.

Another interesting thing about the patients in the hospital at the present time, who are probably going to lose limbs, is that they are all Australians. They worked in the engine room; they had never before been exposed to cold. Of the several cases I saw from the southern waters, they

* Dr. Webster was unable to show his slides because of lighting conditions.

were all colored but one, which again leaves a contrast that is hard to explain.

LIEUT. COMMANDER THOMAS PETERSEN* (Boston, Mass.): The first boy was in the water only fourteen hours. He still has no sensation in his feet, and he still has rather marked atrophy of his hands.

The temperature of the water that day was 28 degrees and the temperature of the air was 27 degrees. This boy had ulcers of both hands and ulcers of both feet, with bleb formation, and he had marked stiffness of his hands at the time. Now he still feels cold severely.

The second boy came from Florida and has lived in Florida all his life. He was in the water only half an hour, but he walked in the snow twenty-five hours without his shoes. He still has no sensation in his feet at all. He had bleb formation on the heels and on the ball of the foot, all of which eventually subsided, but we still are having quite a problem getting his feet back into condition. He said he felt the cold very badly. His hands are all right.

LOUIS G. HERRMANN (Cincinnati, Ohio): Dr. Brownrigg has brought up some most interesting problems in the study of these cases that I think have taught us a great deal. They have worked out the so-called end or late results. I should like to say just a few words about what we might term the early results. In the Ohio Valley, we have a great number of frostbitten extremities because our natives are not protected very well against cold. They get inebriated, fall by the wayside, and the next morning they have frostbitten extremities.

In the last two years we have seen fifty-seven frostbitten extremities. They are not "immersion" this time, but they might very well be the same physiological problem. We have looked upon these as difficulties falling in the realm of pathologic physiology rather than pathology, and I should like to detail what we might consider the pathologic physiology to cold.

In the first place, cold causes constriction of the arterioles in the extremities and if the cold is not too intense and too prolonged, it is followed by hyperactive hyperemia. If the cold continues, the so-called hyperactive hyperemias which are out in the extremity are in a dependent condition where a great deal of blood is trapped in the extremity, a great deal of venous dilitation or capillary paralysis takes place, and the blood remains fluid in these trapped capillary beds for a long period of time.

In the first stage of this, the ischemic stage, the extremity can bear ischemia for a long period of time without undergoing serious changes. It cannot stand anoxemia or anoxia that comes from the capillary stasis, so before very long, diapedesis takes place, blood cells get out in the tissue and you get that particular slate-gray color of the extremity with or without bleb formation; and we believe very definitely that if the therapeutic efforts can be directed toward the prevention of this particular cycle of pathologic

* Dr. Petersen presented two patients who were in wrecks in February, 1942.

physiology, we will be able to prevent many of these serious complications which we cannot cure by any method we know.

In our experience in this particular type of lesion that we see, the numbness of the foot is the result of a chronic anoxemia of the peripheral nerves. It is an ischemic neurotic, if we dare call it that. We see it a great deal, or did see it a great deal until we became very much impressed by the early signs. We treat these people from the very earliest moment we can get them, and they are kept cold, either by the use of ice in ice bags or any method of refrigeration to keep the demand for oxygen down to its lowest level. That, I think, is very important. The next most important thing, I believe—and I think it is borne out by our own cases, which are nothing to compare with Dr. Brownrigg's excellent series—is that if you can keep the blood circulating by any means, by very gentle superficial massage or by perivascular exercise, these serious complications do not result nearly as often.

We have not lost any extremities in the last five years by amputation. We have lost toes. Our degree of frostbite or frozen extremity is nothing like Dr. Brownrigg's presentation, and I am only telling you this because I do believe more careful attention to the early pathologic physiologic signs is extremely important in the prevention of serious complications from the exposure to cold.

EDGAR L. GILCREEST (San Francisco, Calif.): I think an explanation is due you that I, who spent the first half of my life deep down in the heart of Texas, and the latter half looking for a more salubrious climate in California, should discuss the subject of frostbite, but I saw in the Turko-Balkan war in 1913 (the first war where they really had trench warfare) literally hundreds of these Serbian soldiers, who had stood in the trenches in the snow for hours, with what we called trench frostbite. In Britain in 1915, I saw many who had stood for hours in wet trenches with what we called trench feet.

In the last war I happened to be Chairman of the First Corps of Disability of the A. E. F., where hundreds of these soldiers passed through my hands to be returned to the United States.

Previous to our going into the war, we had for several years heard the argentiferous-tongued William Jennings Bryan say we could raise a million men overnight and we should not get particularly exercised over the European war. We did raise a million men overnight, and they were men, not soldiers. The Negroes were sent out of the Deep South to Hoboken, with shoes that were too tight on their feet. They went down into the bottom of the boat. They could not fish and they did the only other thing they knew how to do, slept and shot craps, and they did not need shoes for that. They got to France. It was January. They put on the too-tight shoes and were put on the train, and they rode in trains that had no heat whatever, and their feet began to swell. Dr. Herrmann mentioned to you the

phenomenon that takes place, and the swelling of the feet in those too-tight shoes.

They were taken off the train, put in an ambulance, never walked a foot of soil in France, were brought to the hospital suffering with these various phenomena of vasomotor conditions, and they suffered greatly. They were brought back to the boats weeks or months later in ambulances and brought back to this country. That cost us literally millions of dollars, but the fact is that we saw all these things, whether you call them frostbite, immersion feet or trench feet. The important thing is that in this war we are going to see a great many of the same things; and if we can do anything now to prevent it, it will be a very important thing.

I think we will have no paper during this whole meeting that will be more important than the ones that we have had this morning. In the case of one man, we had to amputate ten of his toes, and walking with your toes gone is like walking on stilts. I think a paper that will bring out what we can do, is very timely; we should educate the public as well as the army general as to what should be done. The whole thing is a vasomotor phenomenon. A great deal of it can be prevented, and I think anything that will bring that out at this time has been a very important contribution.

William Darrach (New York, N. Y.): I shall take one minute and thank the Program Committee for going not only to Boston and the big cities, but when they want something real for going to Newfoundland. Therefore, on the part of the organization I want to thank Dr. Brownrigg for his delightful presentation and for his long trip down here.

Mims Gage (New Orleans, La.): I certainly enjoyed Dr. Brownrigg's paper. Of course, I am down in the South where we do not have very much of this. It is rather interesting to note that we do have it in the Gulf Stream.

I wanted to say a few words about sympathetic block. Dr. Penberthy brought that up, and Dr. Herrmann mentioned the pathologic physiology or the physiologic pathology, and I think in these cases, in the circulation in the foot or extremity, one is dependent upon the other, that is, the arterial, the venous and the lymphatics. As Dr. Herrmann says, once the congestion of the capillaries occurs, the lymphatic flow practically ceases.

Dr. Penberthy showed the excellent results they have from stellate ganglion block, which is so simple that our second and third-year residents do it. It is a simple procedure, and especially in the exudative stage I think it is most enabling, because I have injected about twenty cases of bad infections of the hand in which they had become edematous. We have blocked the stellate ganglion daily for four to five or ten injections, which is very simple and does not produce any damage. You should be careful not to get into the vertebral artery or the dome of the pleura. After the injection in these cases of infected hands which are edematous and sloughish, the patients ask for it because of the relief of pain; and to me the disappearance of the edema in these infections and the rapid healing of the hand is most

dramatic, so much so that I have been afraid to publish it, and I have been doing it for the last six years. I have not published anything because it was quite dramatic and I wanted to wait for a larger series.

I showed Dr. Robert Payne the results and I had a letter telling me that in these infections of the hand with marked edema, and so forth, he had never used anything that gave him more gratifying results; and I think in the exudative stage, in which there is practically complete cessation of capillary pulsation and stoppage of lymph flow, as we have shown in the experimental laboratory, by simple sympathetic block all of this is overcome and the edema rapidly subsides, very much as it does in the thrombophlebitis. For instance, I have seen a case of a very extensive thrombophlebitis of the leg, with the limb swollen from the thigh down to the foot, when it seemed almost as though the skin would rupture from the edema, in which five injections over a period of six days, lumbar sympathetic block with novocaine, brought a normal temperature on the tenth day.

I am sure what others suggested about the application of cold in the first stage is correct, but in the exudative stage I would suggest they use sympathetic block, and I think the results will be most gratifying.

This paper has been of great interest to me. I enjoyed it and I would certainly like to congratulate the essayist.

GARRETT M. BROWNRIGG (closing): I do not think there is very much to add except that I would like to say, in the case of the Gulf Stream, that 50°F. water is still 48° colder than the human body is accustomed to.

HEMATOMAS: EXTRADURAL, SUBDURAL AND SUBCORTICAL

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IN this war period a considerable number of neurosurgeons have entered or will enter the various armed forces. Therefore, general surgeons interested in the surgery of trauma will necessarily have to care for many more cases of craniocerebral injuries.

With this in mind I have prepared the following paper in the hope that it may be of some value to this group of surgeons.

Intracranial operative procedures or explorations are carried out more frequently for blood clots than for any other group of craniocerebral injuries, with the exception of compound and depressed fractures of the skull. In fact, these two groups account for practically all intracranial operations associated with trauma. More often the operation is carried out for a single lesion, but all three varieties of hematoma may be present in the same patient, and they all may be found on one side, one superimposed upon the other. Each group will be considered separately.

EXTRADURAL HEMATOMA

Extradural hematoma is located between the dura and the inner table of the skull. It results most frequently from injury and tearing of the middle meningeal artery, or one or more of its branches. The artery enters the cranial cavity through the foramen spinosum, situated in the base of the skull rather deep in the middle fossa. It passes upward and outward for a short variable distance and divides into two main branches, the anterior and posterior, which supply blood to the major portion of the dura. Tearing through or into this vessel or its branches results in formation of a blood clot between the dura and the inner table of the skull. The size and rapidity of formation of the clot depend upon the site of injury to the vessel and the age of the individual. This lesion occurs more frequently in young adults and patients of middle age, although it may be present at any

age. It is more common in males than in females. Especially in the first decade, the dura is loosely attached to the inner table, and the middle meningeal artery and its branches lie in very shallow grooves, so that an injury to the skull would be less likely to injure the vessel. In the aged group, the dura is more likely to be intimately adherent to the inner table of the skull so that even though a linear fracture might injure the middle meningeal artery or its branches, separation of the dura from the skull is less likely to occur, hence, the paucity of instances of accumulation of blood clot between dura and skull in this group.

A linear fracture of the skull traversing the course of the middle meningeal artery or one or more of its branches is the commonest cause of extradural hematoma, although this lesion also results from more complicated fracture lines including a depressed fragment. In younger adults the fracture line may lacerate some of the smaller branches which do not groove the inner table so decidedly, with the result that the blood clot may be smaller and slower in forming. Doubtless, many instances of small extradural blood clots occur and go unnoticed, because they produce no recognizable signs or symptoms.

The posterior branch is more frequently injured than any other portion of the middle meningeal artery, and occurs as often on one side as the other. The resulting blood clot is biconvex and discoid in shape, similar in shape to the crystallin lens. The size depends upon the amount of injury to the vessel and the ease with which the dura is separated from the inner table of the skull. If the vessel is small and the dura is more firmly fixed, the clot forms more slowly resulting in delay of appearance of the typical signs and symptoms associated with this lesion. When the vessel is completely torn through, with quick outpouring of blood, and when the dura is loosely attached, the clot is larger, is formed more rapidly, and results in death due to the rapid gross compression of the brain in its nonelastic bony case, if the condition is not promptly relieved.

What is stated above refers also to the anterior branch of the artery. The main trunk of the vessel is less frequently injured. One must remember that the trunk of the vessel lies deep within the middle fossa beneath the sphenoidal lobe and nearer to more vital basilar structures of the brain. In this location the clot is more likely to be ovoid, pressing upward beneath the sphenoidal and frontal lobes; and when the vessel is injured at this site, the classical picture is less likely to ensue, due to the absence of hemiparesis or paralysis,

unless the clot rapidly dissects the dura away from the inner table of the skull and extends upward over the motor area.

Extradural hematoma may, but seldom does result from injury of one or more of the larger venous sinuses of the dura, the lateral sinus being more often involved.

Symptoms. The typical syndrome associated with extradural hematoma is pathognomonic. Munro stated, with reference to extradural hematomas: "These are the classical examples and have served as the models upon which the symptomatology of all brain injuries was based."

For practical purposes, all of these clots which come to the attention of the surgeon might be considered acute. I have never had occasion to operate upon a chronic extradural hematoma, unless one of seven or eight days' duration should be so considered. The typical picture is as follows: The patient receives a blow on the head, more frequently on the side than fore and aft, of sufficient force to produce a linear fracture of the skull, more often simple than compounded. Unconsciousness develops, and after a variable interval consciousness returns. The patient may be somewhat hazy and there is usually an interval, the so-called "lucid interval," in which the patient is in control of his senses. He may even telephone from the corner store or some other place, to his family, stating that he has had an accident but feels all right. Following this lucid interval, which may vary from minutes to hours, unconsciousness, perhaps accompanied by hemiparesis or paralysis occurs again, followed by deep coma and deep sterterous breathing. The patient never recovers unless the blood clot is promptly evacuated and bleeding controlled. The pulse is slow and full.

Unfortunately, such a simple picture does not always present itself. Indeed, the symptomatology and diagnosis may be quite obscure. The hemiparesis or paralysis associated with extradural blood clot, when present, is contralateral, and is due to pressure over the motor area. Should the blood clot be low in its position, for example in the middle fossa, without dissecting over the motor area, paresis or paralysis of the opposite side may not be present.

Kennedy and Wortis stated that the presence of a dilated pupil indicates that the clot is on the same side as the dilated pupil. The clot may be bilateral, but this is a rare occurrence. Extradural clot may occur, but seldom does, without fracture.

There may be an underlying homolateral subdural and, or, subcortical blood clot; or an associated contralateral subdural hema-

toma, with or without contrecoup brain damage. The spinal fluid may be clear, pink, or grossly bloody, depending upon the amount of brain injury. Theoretically, the fluid should be clear, but actually this does not always obtain, for the reason that brain laceration in addition to simple compression is often present.

Pathology. The blood clot may vary in size, shape, thickness, color and extent. Its rapid production followed by the acute depression and compression of the brain like a foreign body produces the rapid oncoming symptoms. The hematoma usually consists of solid clots which resemble currant jelly, or they may be as dark as raspberry jelly. Usually the clot is friable, and ordinarily a portion of it is immediately extruded through the burr hole in the skull. According to Vance in his autopsy series, the clot varies in weight from 40 Gm. to 300 Gm., and it may be an inch or more in thickness.

Extradural hematoma complicates craniocerebral injury in about 2 to 5 per cent, and probably is less common than is generally believed. Vance's figures show that a considerable number of cases are never recognized except at autopsy. There were 106 instances of extradural hematoma in his series of 507 autopsy cases, of which sixty-one clots were of sufficient size to produce death through cerebral compression. He stated that the duration of the clinical course was variable, and that over half of the patients died within twenty-four hours. A large proportion were found dead. He advanced the belief that a number, after injury and during lucid intervals, wandered away from the scene of the accident, possibly in fairly good condition, and were overcome by the rapid on-coming stupor and coma, and died without observation. Others were found in deep coma so profound and so prolonged that operation was of no avail. No doubt, in many the condition was confused with the alcoholic state and was not recognized.

Treatment. From the foregoing one can judge that the treatment is surgical demanding immediate removal of the blood clot. If operation is too long delayed, compression of the brain stem takes place to such an extent that the condition is irretrievable. Immediate operation is probably never indicated in cases of craniocerebral injury so positively as in the case of extradural hemorrhage. The urgency of operation is almost as great in some instances as in stab wounds of the heart. In my own experience the presence of the surgeon in the hospital at the time of admission of the patient prevented the loss of life in more than one instance. Had it been necessary to call the surgeon from any distance the period of safety would have passed.

If time permits, the patient should be taken to the *x*-ray room on the way to the operating room so that films, especially lateral films, may be made. In view of the fact that a large percentage of these is associated with linear fractures of the skull, the position of the fracture line, in addition to other clinical evidence, may indicate the point at which the burr hole should first be made. The films can be developed and viewed as wet films while the head is being shaved and the scalp prepared.

Operation is carried out under a local anesthetic. In some instances the stupor or coma may be so deep that no anesthetic will be needed until after evacuation of the clot. The patient may then quickly regain consciousness, and local anesthesia may be required for proper closure of the incision.

The burr hole is made over the site selected through a muscle-splitting vertical or oblique incision, depending upon the position of the clot. The burr hole may have to be made over the postparietal or in the temporal region, and in instances in which one is led to believe the clot is low in position and probably in the middle fossa, the opening should be made low in the squamous portion of the temporal bone just above the zygoma. Osteoplastic flap is mentioned only to be condemned. No time should be wasted in application of numerous artery forceps and skin clips to the scalp. Self-retaining retractors will suffice to provide adequate exposure and control bleeding. When the burr hole is made, a considerable amount of clot will be spontaneously extruded through the opening, if it is properly placed. It is advised that this opening be enlarged so that proper evacuation of the clot and control of hemorrhage may be accomplished without tedious delay, especially when the clot is rather large and thick and in the temporal region. The musculature and scalp will give adequate protection. In instances in which it is not necessary to open the dura, or in which the dura has been opened and closed, and is approximated to the pericranium, the bone will re-form anyhow. In a number of instances evacuation of the clot will suffice. This is done by suction and curettage will not be necessary. The bleeding may have stopped, but if it is mild the bleeding points can readily be controlled by suture ligatures or electrocoagulation.

When the fracture line involves the trunk of the middle meningeal deep in the fossa, the active bleeding may be considerable, and the site of the injury to the vessel may be found with difficulty, if at all. It may be controlled by narrow gauze packing brought out through the lower angle of the incision and removed gradually over a period

of four or five days. Should there be any doubt as to adequate control of bleeding, it is advised that immediate ligation of the external carotid artery be done as indicated in some cases of nose bleed. Ligation will rarely be indicated. The ligation can be done in a few minutes and will add but little risk. Collateral circulation will not prove troublesome.

Should the dura appear "bluish" it should be opened and the subdural clot removed. It is advised that the dura be loosely closed leaving an interval between sutures for the escape of any subdural fluid which might accumulate.

Munro advises opening the dura in all cases, but I see no necessity for this except in the presence of clot or free fluid. With this Coleman agrees. Creation of a dural opening for decompressive effect is seldom indicated. Should a subdural clot be found and removed, a small rubber dam drain is inserted just beneath the dura between sutures, and removed after twenty-four to thirty-six hours. After complete removal of all clots and loose closure of the dura, it is well to fix the elevated dura to the overlying pericranium with sutures in order to obliterate the "dead space" which would otherwise exist. The incision is then closed in layers with interrupted silk sutures. Blood transfusion and infusions may be indicated. In one instance I observed the three types of blood clots through the same incision, the one superimposed on the other, and after removal of all three clots, the patient recovered.

Prognosis. In uncomplicated cases, rapid and complete recovery may be expected. The patient may "wake up" on the table and have to be restrained by assistants and belts. On the other hand, the tremendous degree of compression may be so great and prolonged that the patient will not recover regardless of the adequacy of the operation. One must always consider the possibility of a concurrent injury or lesion, and should bear in mind that the blood clot may be bilateral, especially in circumferential linear fractures. There may be a subdural clot on the opposite side, or gross damage by contrecoup. If the patient's condition does not show the expected improvement following evacuation of the extradural clot, one should not hesitate to make a burr hole on the opposite side. It is humiliating to see the patient lapse into coma followed by death, and have autopsy reveal a large clot on the opposite side. It is even more humiliating at autopsy to find that the burr hole was placed a short distance from the margin of the blood clot without localizing it. Probably most of us have had this experience.

In conclusion, practically all patients with uncomplicated extradural blood clots overlying the cerebrum should promptly recover if the operation is performed at the proper time, the blood clot is completely removed, and the bleeding adequately controlled. Unfortunately, many cases are seen too late, and others show severe complications.

With reference to the extradural clot of the posterior fossa, a few words might be said. Here the clot most usually results from injury of the lateral sinus or one of its tributaries, although it could be arterial in origin. Smaller clots may prove to be more quickly disastrous on account of the small space involved in the posterior fossa, and the fact that the compression involves the more vital centers. Fortunately, this group is very small. It goes without saying that evacuation of the clot and control of bleeding should be carried out immediately, and even so the prognosis is not favorable.

SUBDURAL HEMATOMA

Although the condition was described by Virchow, in 1857, and termed "pachymeningitis hemorrhagica interna," and four typical chronic cases were reported in their entirety by Trotter, in 1914, subdural hematoma as an entity was not properly recognized until after the report of Putnam and Cushing, in 1925. When one considers the fact that the lesion frequently results from the mildest kind of trauma or a bump on the head, one must concede that this lesion most assuredly has occurred since the beginning of mankind. It is almost unbelievable with the other advances in modern surgery, that this condition evaded recognition by the medical profession until recent times. Only a few articles on this subject appeared in the literature in the late 1920's after the paper by Putnam and Cushing. However, since 1930 many splendid articles and papers have made their appearance. Now, the "typical" subdural hematoma is recognized by neurologists and neurosurgeons as an entity, just as is the extradural clot.

The occurrence of this lesion seems to be frequent in recent times contrasted with its almost complete lack of recognition prior to 1925. During our intern days in 1909 to 1915 we heard of the diagnosis of "pachymeningitis hemorrhagica interna" made at autopsy by Doctors Charles Norris and Otto Schultz, but further than the diagnosis the condition was not understood. It was not known as a "subdural blood clot." We were completely ignorant of the nature of

this lesion at that time. Deaths resulting from this type of clot evidently were ascribed to a "cerebral attack," apoplexy, or some other intracranial episode which was not understood or appreciated. Even in this day and time, most cases of chronic subdural hematoma are referred by the patient's physician to the neurologist or neurosurgeon with the probable diagnosis of "brain tumor."

General surgeons who may be most proficient in all their other undertakings, may not be sufficiently aware of the presence of a subdural clot which could readily be evacuated by any one of them, were the condition recognized. Considering the fact that these accidents occur over such a widespread area, and the paucity of neurosurgeons, it behooves the general surgeon, especially those who devote much of their time to traumatic surgery, to pay more attention to the trivial accidents that result in this condition which may be so readily relieved by simple trephine in many instances. For this reason, emphasis on this subject is made.

Subdural hematoma should be considered as "acute" or "chronic" hematoma.

Acute Subdural Hematoma. It is well understood that if an acute hematoma is not removed, and if the patient lives sufficiently long, the lesion becomes "chronic"; that is, the chronic lesion is merely a later stage of the acute condition. No time limit has been set to indicate the termination of the acute stage and beginning of the chronic stage. Kennedy and Wortis, in their paper, included in the acute group only cases occurring within twenty-one days.

From the practical standpoint, acute cases should be subdivided into two main groups: First, those associated with massive and gross trauma of the skull and brain, in which the subdural clot may or does play a minor rôle; and second, those cases which usually follow minor trauma to the head, with no injury to the skull, and but little injury to the brain and its coverings. In the second group the subdural clot plays a major rôle.

In the first group most of the patients will die with or without treatment. Even when the clot is removed the mortality is very high. Consideration of the treatment and management of this group does not fall within the scope of this paper.

In the second group it is believed that a "simple" uncomplicated acute subdural blood clot results in most instances from a mild blow, probably on the anterior or posterior aspect or pole of the head. The blow or trauma may be so mild that it will not be remembered by the patient, or the patient when examined may be in such deep stupor or

coma that no history of a blow can be obtained. The blow may be so trivial that no evidence of it can be found by examining the scalp, and it is usually not associated with fracture of the skull. Most writers believe that the clot forms as a result of tearing or rupture of the cortical veins in the free subdural space after their coalescence and before their entrance to the major or venous sinuses of the dura. It is believed that the trunks of the cortical veins suspended in the subdural space between the cortex and the dura are torn through by propulsion of the brain bulk forward or backward, after sudden arrest of motion of the skull by the impeding object against which the skull is struck. In most instances in which history can be obtained it will be determined that the "head was struck against something" rather than the "head was struck *by* something"; i.e., the head was in motion. Typical examples are cases in which the patient stoops over or leans backward and strikes the head against a table, piano, bathtub, etc. In all these instances the motion of the head is suddenly arrested, while that of the brain continues. The force and excursion are sufficient to tear the cortical vein or trunks, without necessary contrecoup damage to the poles of the brain. One might crudely but graphically compare this picture with that of a person standing in a wagon-bed, driving a team of horses at full tilt. Should the horses stop suddenly and unexpectely, the driver most likely would be propelled forward against the wagon-bed. The shift of the brain in its case is greater in the fore and aft directions than from side-to-side, due to the fact that the falx cerebri acts as a stabilizer in the lateral direction, as well as the fact that the brain bulk is greater in the anteroposterior diameter. The greater width and fixation of the falx at its posterior extremity may account for the fact that the clot usually extends more over the frontal than the occipital pole of the cerebrum.

In addition to the assigned type of trauma, there must certainly also be some predisposing cause, such as short trunks of the cortical veins, weakness and friability of the venous wall, etc. No doubt every living person has struck his head, not once but many times, with various degrees of violence, yet only a few persons suffer from formation of a subdural clot. In fact, a clot may form from spontaneous rupture of a vein in the subdural space without evident trauma, a fact also recognized by Virchow. Halpern reported a case in which a cerebral aneurysm ruptured at the point where the internal carotid passes through the subdural into the subarachnoid space to continue as the left middle cerebral artery. The author observed the same

condition in a patient about four months ago. In this patient the pupil alternated several times between normal size and marked dilatation. The aneurysm was no larger than a split pea.

A large subdural clot may also result from injury of the major venous sinuses and may terminate in early death. It may also be the result of complete division of the middle meningeal artery with a markedly adherent, lacerated dura. The proximal end of the artery may project through the rent in the dura and quickly pour its full volume of blood into the subdural space. If it is not removed immediately, death promptly ensues.

When the vein is torn, free bleeding takes place over the cortex, usually the cerebral, with varying degrees of rapidity and volume. Should the anterior veins beneath the frontal or sphenoidal poles be torn, the clot may extend downward beneath these lobes, or upward and backward over them, or both. Or, the injured vein may be posterior in position, with the major portion of the clot in this vicinity. More commonly one finds the clot extending over the cortex occupying practically the entire length of the cerebrum from pole to pole, in an anteroposterior direction, and from near the midline downward below the upper portion of the squama. Therefore, its horizontal length is usually greater than the vertical, and thus differs from the discoid or ovoid form of extradural clots. The clot is thickest in its central portion, and biconvex in shape, the external surface being greater in convexity. It may be an inch or more in thickness. The clot may be solid, having the typical dark blue-black color of clotted venous blood if seen early after its formation, if only venous damage has occurred. Frequently, the pia-arachnoid will have been torn, in which instance the clot will be more fluid in character. In fact, the bulk of the mass consists of dark bloody fluid, decidedly more fluid than clot, and can be readily evacuated through a small incision in the dura.

The cerebral mass is displaced downward and toward the opposite side by the clot in unilateral lesions; if bilateral, downward only for the most part. The associated symptomatology is due to the compression and displacement of the cerebrum rather than to the internal loss of blood *per se*.

The pathology, including composition, organization and partial liquefaction of the clots has been fully and adequately described by Munro and Merritt, Putnam, Gardner, Zollinger, and other writers, and will not be considered in this paper, primarily intended for the practical use of the surgeon.

Symptoms and Signs. One could expect that a displacing lesion of such large proportions would be followed by bizarre signs and symptoms. This may or may not be the case. The lack of signs and symptoms may be marked. There may be only mild headache. Should this increase, a physician may be consulted, and after a cursory examination the patient may be dismissed with a prescription for medication for the relief of headache.

This picture in cases of simple uncomplicated acute hematoma may prevail from hours to several days. Sooner or later, however, certain signs and symptoms will appear, partly subside or disappear, and then recur. Although there is no pathognomonic syndrome, as is present in a typical case of extradural hematoma, it is believed that the effect on consciousness in the "in and out" picture, as we call it, is one of the most constant findings and leads one to suspect promptly the presence of a subdural clot. More often one may obtain the history of some injury to the head from the patient, family or witnesses. There may be a period of loss of consciousness from two to twenty-four hours, headaches, and irritability, from all of which he may apparently recover, with a lucid interval of three days to several weeks or months. On the other hand, drowziness may progress into deep stupor and coma, from which the patient may not recover.

All varieties of changes in the reflexes and peripheral pareses or paralyses have been observed. Unilateral central type facial weakness is common. The hemiparesis or paralysis, when present, may be home- or contralateral, possibly about equally divided, but in my experience, more often on the homolateral side. To my chagrin, in the earlier days before this fact was known to me, I had a disappointment similar to that of Grant, Gross and others, in that only one exploratory burr hole was made, and that on the contralateral side. This patient had been walking about for five days after a mild blow on his head, and came to the hospital under his own power. At autopsy an enormous clot was found on the homolateral side.

The pupil may be dilated and fixed without ptosis of the eyelid. Some writers state that the dilated pupil is found as often on one side as the other, with reference to the location of the clot. At Bellevue Hospital we noted that it was more common on the homolateral side, and was one of the chief indications for selection of the site of the first burr hole.

The cerebrospinal fluid may be clear and of normal contents, if there is no laceration of the brain, but may be xanthochromic, pink

or grossly bloody depending upon the amount and position of the brain damage or laceration. It usually shows considerable elevation of pressure by the manometer in this stage, but the pressure may be within normal range. The pulse may be slow, normal or increased in rate, and may vary in rate and volume even while the hand is on the pulse. Leukocytosis is usually observed due to the presence of the blood clot or blood in the cerebrospinal fluid or both. The temperature is not markedly elevated in the simple uncomplicated cases. Should it be above 103°F., it is due most likely to a concomitant infection or severe brain damage, and the outlook is poor. Papilledema is not commonly found in the early stage, but engorgement of the retinal veins may be observed. At the time of the examination in the hospital the patient may be conscious, stuporous or in deep coma.

Diagnosis. The diagnosis may not be made readily on admission. Dehydration therapy is usually carried out in the time consumed in making the diagnosis. One should strongly suspect the presence of an uncomplicated acute subdural hematoma with the following findings: history of slight injury to the forehead or back of head followed by a period of unconsciousness lasting from a few minutes to hours; stupor, or loss of consciousness, especially of the "in and out" type; a lucid interval of several hours or days; negative *x*-ray films of the skull; hemiparesis; dilatation and fixation of one pupil, with lumbar puncture revealing cerebrospinal fluid clear, xanthochromic or pink, and usually under increased pressure. Other signs and symptoms may be of additional value, but those enumerated constitute sufficient evidence to warrant strong suspicion of the presence of an acute subdural hematoma.

If an electro-encephalographic apparatus is available, and the patient's condition permits, electro-encephalograms may prove of great value, not only in determining the presence, but also the lateralization of the lesion. This coincides with the opinion of Schwab. One might expect that air studies would reveal marked distortion and displacement of the ventricles, but on the whole, air studies have been of little value. Substitution of air by the lumbar route, even in the presence of low or normal manometric pressure, might prove disastrous due to jamming of the conus, or herniation of a part of the cerebrum through the opening in the tentorium. Manometric pressure of the spinal fluid is not always the same as that of the intracranial pressure. Ventriculograms will be precluded in most instances for the reason that the clot will probably be found

when bilateral burr holes are made. Should no clot be located when the holes are made, ventriculograms are then indicated and should be made at this time.

Treatment. Except for supportive treatment in addition to dehydration previous to operation, the most essential thing is removal of the clot. Operative procedure should be carried out under local anesthesia. The head should be placed in position and draped so that bilateral exposure is obtained. In every instance, with no exception, bilateral burr holes should be made, for the reason that the subdural clot is frequently bilateral. Ira Cohen stated that, if the clot is unilateral, he would prefer making the burr hole on the wrong side first, so that if no clot is found, he will be sure to investigate the opposite side. Some prefer the temporal approach, and some the postparietal. Multiple burr holes may be necessary in the localization and removal of the clot. At any rate, the burr hole is made through an incision not more than 1¼ inches long, and it is well to make both openings before the dura is incised. On the side of the clot the dura will appear discolored—blue, or bluish-green. When the dura is incised, a "liquid clot" will evacuate itself without difficulty; in fact, the fluid pours out in varying amounts with pulsation. Some surgeons advise irrigation of the subdural space with saline to facilitate removal of the blood elements. Others advise strongly against it in the belief that it increases edema of the brain. A rubber dam drain should be inserted into the subdural space through the small incision, and brought out through the lower angle of the incision in the scalp, and the scalp closed. The drain is partly removed in twenty-four hours, and completely removed after forty-eight hours. This simple operative procedure suffices with liquid clots.

Should the clot be solid, consisting of typical coagulated blood, it is advised that the opening on the side of the clot be enlarged quickly with rongeurs to the size, approximately, of a silver dollar and the incision in the dura be lengthened. By so doing, the solid clot can be readily and thoroughly removed by suction under direct vision. Passing the suction tip in all directions without direct vision may result in tearing into another vein, with re-formation of the clot. Therefore, this should be avoided. When the clot has been evacuated, it may be observed that the cortex will be depressed, although the convolutions otherwise may appear normal. The surface of the cortex may be stained by the hematogenous elements. The depression in the cortex may disappear immediately. When the depressed cortex becomes vigorously elevated, marked increase of the intraventricular

cavity can be suspected. After complete evacuation of the clot, the dura is loosely closed with a rubber dam drain inserted just within the subdural space. This is removed during the next twenty-four hours.

Munro rightly states that the postoperative care is very important. In all instances we have performed postoperative lumbar punctures, sometimes daily, until the patient is "out of the woods." (Previous to operation a lumbar puncture is done only for diagnostic purposes.) Dehydration may be done, but in the majority of instances will not be found necessary. Reaccumulation of the clot may occur, and one must be on the alert for this possibility. It is not a common occurrence.

Prognosis. In the majority of patients operated upon after the seventh day, recovery should be expected, and is usually without untoward event. The high mortality reported by Kennedy and Wortis, and various other writers, is due to the fact that the first group of cases, mentioned but not considered in this paper, is included. Kennedy and Wortis reported 43.7 per cent in the operative mortality in thirty-two cases from the Neurological Service of Bellevue Hospital. Thirty-one patients died without operation. Other writers report an equal or higher mortality when cases of gross skull and brain damage are included.

Frank Turney recently informed me that the approximate operative mortality rate at Kings County Hospital was as follows: During the first twenty-four hours, 88 per cent; from first to third day, 70 per cent; from third to seventh day, 42 per cent; from seventh to eleventh day, 15 per cent; from eleventh to fourteenth day, between 8 and 10 per cent; and from fourteenth to twenty-first day, 12 per cent.

Francis Echlin informed me that during the approximate eighteen months in which he has been in charge of the neurosurgery in the Bellevue Psychopathic Hospital, they operated upon about eighty patients with subdural hematomas with the following results: Very few were operated upon during the first twenty-four hours. Most of them were operated upon after the third day, especially after the seventh day. Practically all those operated upon in the first twenty-four hours died; while the majority of those operated upon after the fifth day recovered, provided they were not in prolonged, deep coma. From these figures and statements it can be concluded that the majority of patients operated upon should recover, provided there is no associated gross damage of the skull and brain, or deep, prolonged

coma. In the Kings County Hospital series, the slight increase in death rate between the fourteen and twenty-one days was due to the concomitant infections, pulmonary involvement and general debility.

Chronic Subdural Hematoma. If an acute subdural hematoma is not recognized and removed, and the patient survives, it becomes chronic. It may be present for weeks or years without being recognized. The outer and inner membranes may be calcified with a liquid central portion. Lillie reported removal of a completely calcified lesion over twenty years after the injury.

Some patients may show practically no signs or symptoms, and certainly none sufficient to justify operative interference. On the other hand, it is believed that a history will be obtained either from the patient or members of the family which will cause one to suspect the presence of a chronic subdural clot. The symptoms of irritability, lack of interest, inability to work or concentrate will, most likely, be brought out by proper questioning. His friends will have noticed that the patient has changed, that he does not behave normally, and that he acts queerly.

The one outstanding feature in many cases is headache, intermittent, or continuous, sometimes mild, but in many cases very severe and constant, necessitating cessation of work. Nausea and vomiting may accompany the headache. It may be unilateral, but is often generalized, and is of a persistent, throbbing character. Coleman stated that in over half of his series of cases, headache began immediately or within two days after injury. Delusional and maniacal manifestations may be present, and some patients have been confined to institutions for the insane.

History of trivial injury may be obtained. Patients in stupor, who cannot give a coherent history, may recall the injury a few hours or days after evacuation of the clot. There may not be any period of unconsciousness. Stupor or unconsciousness alternating with consciousness is not uncommon, and constitute the "in and out" state so often spoken of by Foster Kennedy. The gradual passing from drowziness to stupor, loss of consciousness deepening into coma, may be observed, and bodes ill unless the clot is immediately removed. This picture is most likely to occur during the first three or four months, but may occur in patients with older clots.

Hemiparesis is present in about 50 per cent of the group, and is more often homolateral in the early cases, and contralateral in the late or very chronic stage. This occurrence is explained by the fact that the lesion in the earlier stage is a fairly rapidly expanding soft

lesion which presses the opposite hemisphere against the skull, flattening the convolutions, or the brain stem against the opposite sharp margin in the incisura in the tentorium, or both. After many months or years the brain is not so likely to be affected in this manner. The clot remains more or less as a simple foreign body, like a tumor, locally compressing and displacing the underlying motor area, thereby producing contralateral motor weakness similar to that of a tumor.

There may be homo- or contralateral dilatation of the pupil, but this is not so frequently seen as in the acute cases. Papilledema may be very marked or there may be no changes in the disk. Most of the cases in this group are referred to the neurologists and neurosurgeons as "brain tumor suspects," and the clot may be found when the burr holes are made for ventriculography in the localization of the suspected tumor. The diagnosis of "allergy of the brain" has been made. Frequently, the positive diagnosis is established after the burr holes are made.

Treatment. As in the case of acute subdural hematoma, the treatment is surgical, and consists of removal of the clot. Local anesthesia is used. In all instances the clot should be removed in its entirety if the condition of the patient will permit. In a few cases in which the condition of the patient is very poor, it is advised that the liquid portion of the clot be removed through a burr hole with no loss of time, as suggested by Coleman, wait for improvement, and remove the clot in its entirety at a later date. It is not believed that chronic organized clots can be successfully removed through a burr hole, nor is it believed advisable that only the liquid portion should be removed, leaving the bulky membrane in position. Bilateral burr holes in the upper temporoparietal region should always be made for the purpose of making a diagnosis, because a number of these lesions are bilateral. The clots may be of equal or unequal size. Burr holes are preferred to air studies in the majority of cases. The lesion most likely will be found and can be removed at the same time. If the clot is of fairly recent formation, so that the outer membrane strips easily from the dura, it can be removed more readily and completely after enlarging the burr hole, as is done for acute subdural hematoma. Coleman prefers this procedure to a bone flap.

In the very chronic clots which have existed for many months or years, the dura may even then appear bluish or greenish-blue when viewed through the burr hole, or it may not be discolored. In the latter, the dura offers a wood-like resistance when cut, and only after

incision of the outer thickened portion of the dura, will the dark color of the inner portion be seen, indicating the true nature of the underlying clot. In such cases the outer membrane will have become so firmly organized and fixed to the dura that it may be removed with difficulty even after a flap has been turned.

This type of clot cannot be properly removed through a simple burr hole, and since the bony opening would have to be enlarged to undesirable proportions, I have preferred making a bone flap through a straight or slightly curved horizontal incision. The incision is made in the direction of the branches of the supra-orbital nerve, which extend far backward toward the occiput, thereby resulting in a smaller, postoperative anesthetic area of the scalp. The galea and scalp are reflected from the pericranium and outer fascia of the temporal muscle exposing an area larger than the palm of the hand. The bone flap, turned downward and hinging on the temporal muscle, is longer in the horizontal than the vertical direction, being in the proportion of about three inches to two inches. The bone flap is so fashioned for the reason that the underlying firm clot is longer in the anteroposterior than in the vertical direction. After the dural flap is made, the outer membrane is incised and the fluid portion of the clot is evacuated by suction. A considerable amount of the inner, soft grumous and friable portion of the clot is also removed by suction. The bulk of the mass is thus decreased, but there is no tendency whatsoever to spontaneous separation of the thick, tough, adherent outer membrane from the dura. With care and a little time the clot can be stripped away from the dura. Sometimes considerable force may be required, comparable to that exerted to strip the lining from a chicken gizzard. The brain spoon is an effectual instrument for this purpose. One should be especially careful in freeing the peripheral margins, otherwise tearing of a cortical vein or the pia-arachnoid and cortex may occur. This portion of the clot is often quite adherent to the pia-arachnoid. Small pieces of clot should be left attached rather than cause damage to the underlying veins and cortex by complete removal. Recurrence of the clot will thus be prevented. Removal of the inner membrane occasions no difficulty due to its very loose attachment.

After removal of the clot the cortex fails to expand immediately in most cases of both acute and chronic subdural hematoma, as stated by Coleman, but contrary to the statements of some writers. I am unable to explain this phenomenon. A rubber dam drain is inserted beneath the dura, as described, and is brought out through

a burr hole at the upper posterior portion of the flap. After loose closure of the dura, the bone flap is replaced and fixed with three rustless steel wire sutures passed through small drill holes. The scalp incision is quickly closed in two layers. The drain is slowly removed and shortened over a period of three or four days, during which time a considerable amount of fluids may be given with the patient in the prone position, to aid in expansion of the brain.

Although the postoperative course may be uneventful, some patients may have a very stormy period, even after a few days of well-being. They may require dehydration. If manometric readings show increase of pressure from 190 to 250, therapeutic lumbar punctures are done, daily if necessary. Curiously, the contralateral hemiparesis may increase several days after removal of the clot, even amounting to hemiparalysis for a variable period. Since the drain is removed over a period of several days, reaccumulation of fluid in the subdural space is not expected. I am not able to give definite information regarding this possibility for the reason that the flap has never been re-elevated. It is believed that the increase in paralysis, persistent for several days or weeks, may be due to dislocation of the motor cortex and the connecting motor fibers, resulting from the expansion and elevation of the depressed cortex. The cortex has been depressed so long that it may have become more or less adjusted to the compression. When this is suddenly relieved, the depression of the cortex should disappear sooner or later. It is quite conceivable that the change in the relationship of the motor area and motor fibers could be as great following expansion as compression of the cortex. In other words, dysfunction of the motor area can be caused by the marked displacement due either to its expansion or compression. Increase in the edema of the cortex probably accompanies the expansion and may account to some degree for the increase in the paralysis. During this period, convulsions involving the paralyzed side are prone to occur.

Prognosis. A good, and frequently complete recovery can be expected in the vast majority of these cases, regardless of the rather stormy periods through which some of them pass. If operation is performed on a patient who has been in deep coma twelve hours or more, the result will probably be fatal. Should coma be of short duration, or only deep stupor, recovery can be expected. Deep coma caused by chronic subdural clots, induces or produces irretrievable changes in the vital centers in a relatively short time compared with that associated with tumor. It is believed to be due to a sudden and

violent increase in intracranial pressure. One may note a similarity to the mechanics and sequelas associated with gross hemorrhage about or in a tumor. If the coma develops shortly after admission, operation will likely prove effective if carried out immediately. Sachs stated that all patients in his series operated upon in deep coma, died; all others survived. The other chief causes of death are infection, pulmonary complications, and general debility, especially in older people. On the whole, satisfactory results may be expected in this group of cases. Great satisfaction is experienced in the return of these patients to their homes and livelihood, most of whom would eventually die as a result of the clot, be relegated to a life of uselessness or become inmates of an insane asylum.

Subdural Sacculated Collections of Fluid. A collection of fluid in the subdural space has been known by various names, e.g., "fluid or liquid-hematoma," "hygroma," "hydroma," etc. It seems that one name is as applicable as another, and probably that "hematoma" is the least acceptable. "Hematoma" merely means a "blood clot," and by no stretch of the imagination can the lesion under consideration be a clot. A clot consists of both solid and liquid portions, but the solid predominates. I do not consider a subdural collection of cerebrospinal fluid, originally mixed with a small amount of blood, a hematoma, anymore than intraventricular or sacculated intraspinal fluid mixed with a small amount of blood. The fluid may be colorless, xanthochromic, pink, or somewhat red. Regardless of the terms used, all writers seem to agree as to the nature, symptoms and signs of the lesion, and since they are very similar to those of subdural hematoma, this lesion is briefly discussed. It is supposed to result from a tear of the pia-arachnoid, with leakage and collection of cerebrospinal fluid in the subdural space. However, one cannot account in this manner for similar collections of non-infected fluid associated with otogenic conditions and osteomyelitis of the skull. The proportion of hematogenous elements is small.

The collection of fluid may be unilateral or bilateral, comparable in size and extent to a subdural clot. The treatment is simple and consists of removal of the fluid through a burr hole, preferably placed over the posterior pole of the collection of fluid to favor more complete evacuation in the prone position. A small rubber dam drain is used and removed after forty-eight hours.

This condition should always be borne in mind in dealing with old head injuries regardless of whether they are compensation cases or not. These patients should not be glibly or lightly dismissed, tagged

as "post-traumatic neurosis." This term is so easy to use. My attention was first called to the condition in 1923 before the appearance of the paper by Naffziger. An ex-soldier had received a glancing gunshot wound of the anterior portion of his head several months previous to my examination. The bullet struck the outer table of the skull just within the hair-line, without fracture of the skull. It was not known whether the patient fell and struck his head. The wound healed leaving a small oval bald spot about an inch long, about three-quarters of an inch left of the midline. He complained bitterly of headache and tried to jump out of the window to commit suicide. He had no other symptom or sign on neurological examination. The attending physician called him a "neurotic" and a "nut," and paid no attention to him. Several months after listening to his complaints, and without doing anything for him, the physician prevailed upon me much against my better judgment at the time, to make a "psychologic" burr hole directly over the site of the bald spot. I did so, using local anesthesia. When the dura was incised, an unbelievable amount of clear, colorless fluid poured out through the dural incision. After some of the fluid had escaped, the patient said: "Doctor, my headache is going," and he had no idea as to what I was doing. I increased the size of the burr hole with rongeurs and lengthened the incision in the dura so as to get a peek beneath it. After a great amount of fluid had escaped, I was truly amazed at the considerable capacity of the empty subdural space. The cortex remained depressed and showed very little evidence of becoming elevated. The patient's head was turned to the left side so as to lower the level of the opening, and he was placed in Trendelenburg position. The change in position of the head was followed by escape of much more fluid. After the patient had been returned to the original position, the incredibly commodious subdural space was gazed at. Reasons for the existence of this collection of fluid were not apparent at the time. Upon conclusion of the operation, the patient stated that he had no headache for the first time in months, and that he felt quite well. He made an uneventful recovery, to the surprise and chagrin of the attendants and myself. As a matter of curiosity and interest, he was kept on the ward for a period of at least eight months, during which time he made no complaints. Since this episode I have seen a number of similar cases. It behooves the medical profession to consider well the likelihood or possibility of this lesion before the diagnosis of "post-traumatic syndrome" is made.

Subdural Collections of Fluids in Infants. This condition is usually mistaken for hydrocephalus, brain tumor, some kind of birth injury or congenital abnormality. The head enlarges slowly and gradually, differing from the more rapidly enlarging head in true obstructive hydrocephalus. The veins of the scalp are dilated and prominent. Although the baby may take its feedings, it may refuse them from time to time without apparent cause. It has bouts of fretfulness. Accompanying the slow enlargement of the head, changes of the fundi will be observed. Hemorrhages are not uncommon, and choking of the disks in varying degrees will be present. Convulsions occur and may or may not be associated with hemiparesis. The lesion is frequently bilateral.

The diagnosis can be established by puncture of the subdural space through the lateral recesses of the fontanel. Repeated punctures for therapeutic reasons should not be done because they are ineffective.

Treatment. Munro advises removal of the fluid and a portion of the membrane and states that "it does no harm to leave some of the membrane in place." He and others advocate the subtemporal approach. I prefer making a small flap of bone about 2 inches in diameter, in the temporoparietal region, turned down through a three-inch straight incision similar to that for subtemporal decompression. Local anesthesia is used. The scalp flaps are readily reflected from the pericranium and outer temporal fascia and are held with self-retaining retractors which afford adequate exposure and control of bleeding from the scalp. Two burr holes are made, one anteriorly and the other posteriorly in the upper part of the exposure, about 1½ or 1¾ inches apart. The skull between the burr holes is cut on a bevel with the Gigli saw. It is quite thin and cuts readily. The remainder of the flap is cut with an especially small deVilbiss bone forceps and the flap is hinged below on the temporal muscle. All of this can be done in five or six minutes on account of the thinness of the skull. Bleeding is minimal. A similar, but inverted flap, is made in the dura. When the dural flap is reflected upward, one sees a perfectly transparent corneal-like membrane, with a slight pinkish or flesh-colored tint. One can view the depressed surface of the cortex through the membrane and its enclosed fluid as clearly as one sees the bottom of the ocean through a glass-bottomed boat in the Bahamas. It makes a pretty and interesting picture. The membrane offers a surprising degree of resistance to the puncturing knife, considering the appearance of the cellophane-like structure. When it

is incised, the fluid escapes freely and the sac collapses. The fluid may be colorless or faintly yellow. The membrane is grasped with a forceps and a general drag is made to determine its points of attachment to the pia-arachnoid. The lesion occupies a position similar to that of a subdural hematoma. Most of the membrane can be removed. Force should not be used lest a cortical vein or the pia-arachnoid be torn. A small piece of the membrane left attached here and there does no harm, and laceration of the vein of the cortex should be studiously avoided. After removal of the fluid and the membrane, the depressed cortex does not become elevated, even when a similar lesion is known to exist on the opposite side. In this respect the cortex behaves in a similar manner to that described in the cases of old, chronic subdural hematoma. Therefore, a small Penrose rubber dam drain is fixed between two of the dural sutures along the posterior limb with the distal end protruding about ¼ inch beneath the dura into the "dead space." The proximal end is brought out through a small stab wound in the posterior scalp flap and fixed to the scalp with a silk suture to prevent possible displacement. The bone flap is returned to its position and fixed with one rustless steel wire ligature, passed through small drill holes. Thus, no gross defect in the skull remains. This procedure is preferred, since no decompression is indicated or desired. The scalp incision is readily closed in two layers. The drain is removed on the second and third days.

If the lesion is unilateral, the operation suffices. If bilateral, I prefer waiting two and one-half weeks, after which time a similar procedure is carried out on the opposite side. It is believed that better results will be obtained through employment of a small bone flap and the two-stage procedure in bilateral lesions. The bony defect is unnecessary and these small infants will not stand "too much operating." The results should be and are good in most instances.

SUBCORTICAL HEMATOMA

Subcortical hematoma may be traumatic or spontaneous. In those resulting from trauma, the blow may be severe and crushing, or fairly mild in character. The former, resulting from severe crushing blows, do not come within the scope of this paper. Only circumscribed lesions, whose bulk is entirely, or for the most part within the brain substance will be considered. The clot may be located in any part of the cerebral hemisphere and may destroy vital centers immediately, or they may remain comparatively quiescent and

unlocalized. They may occur singly or in patients with extradural or subdural clot, or both. About three years ago I operated upon a female patient who presented all three types of clot, one superimposed upon the other. The operation was carried out on the ninth day. The patient was in a stuporous condition accompanied by left hemiparesis. A rather long horizontal linear fracture extended through the midtemporal region of the right side, turned downward and bifurcated in the middle fossa. Therefore, the first burr hole was made directly over the fracture line. An ovoid extradural hematoma was removed from a low position over the sphenoidal lobe, rather deep in the middle fossa. Following its removal it was observed that the dura was blue, and when incised a typical, partly solid subdural clot was found and removed. It was then noticed that a small area of the surface of a subcortical clot presented itself on the surface of the temporal lobe. This subcortical clot was removed and proved to be solid and somewhat larger than a golf ball. It is probable that a laceration to the temporal lobe, with bleeding from the depth, occurred, and that both subcortical and subdural clots originated from this source. She made an uneventful recovery and at the present time is carrying on in her profession as a nurse.

For our purpose subcortical hematomas of spontaneous origin can be grouped in two classes, the one so well described by Craig, and the other seen in apoplexy, resulting from rupture of the lenticular artery. The pathology of the two lesions is so well understood that it will not be discussed.

Treatment. When the presence and localization of the clot in the first group is strongly suspected, a burr hole should be made in the overlying bone and a brain cannula of large caliber with multiple openings should be directed toward the position of the clot. When entered, a definite lack of resistance will be met. A major and frequently sufficient amount of the clot can be aspirated by suction, the cannula being used as a suction tip. In some instances the nature of the lesion may be determined while exploring for a brain tumor. If so, it should be evacuated without drainage.

Dr. Wingebach and I have aspirated a clot in several cases of apoplexy or stroke and without doubt saved the lives of patients who surely would have died. One appeared to be breathing his last gasp, and it was doubtful if he could be placed in an ambulance and taken alive to the hospital. When placed on the table he was in deep coma, with Cheynes-Stokes respiration. An enormous amount of clot, the color of blackberry jelly, was removed by suction and was followed by improvement in his condition. His first few weeks in the hospital

were rather stormy, and during this time, repeated tapping of the area was done. At each successive aspiration, a smaller amount of old blood was removed. His recovery was rather prolonged, but his life was saved. He now walks with spastic gait, but his mentality is clear.

From our experience with these cases we advise removal of a considerable portion of the clot in a selected group of cases of apoplexy, at first comprising otherwise hopeless patients. It is quite possible that the usefulness of this procedure may be extended and a considerable portion of the clot may be aspirated in other cases of apoplexy. Once the hemorrhage which occurs from the ruptured vessel has become arrested, the ends of the vessel become thrombosed. It is doubtful that recurrent bleeding would follow aspiration of a major portion of the clot. The syndrome is produced by sudden invasion of the brain substance by the rapidly forming clot, frequently of enormous proportions. The lessening of the bulk of these clots by aspiration might be as imperative as the removal of large extradural hematomas. The clot is always in the same position, varying only in size. A major portion of it can be aspirated through a burr hole made in the mid postparietal region.

To those surgeons particularly interested in the subject of intracranial hematomas, Munro's monograph on craniocerebral injuries, Vance's report of autopsies performed at Bellevue Hospital, and the splendid papers by Putnam and Cushing, Coleman, Craig, Dowman, Gardner, Grant, Gross, Kaplan, Kennedy and Wortis, McKenzie, Naffziger, and many other writers, which have appeared in the literature in the last decade, are recommended.

CONCLUSIONS

In this paper an attempt is made to present the subject of intracranial hematomas or blood clots and the allied conditions from a practical viewpoint, bearing in mind the general surgeons interrested in traumatic surgery, who in this time, because of their availability and the depletion of neurosurgeons will have to care for many of these patients. Very few, if any, original thoughts have been presented. No statistical or theoretical studies have been made.

Hematomas comprise one of the two major groups of intracranial lesions which can and should more often be relieved by timely and proper surgical intervention. Hemorrhage into the subarachnoid space and that associated with aneurysms have not been considered.

If this attempt may prove of any aid to the author and his colleagues in the restoration of these seriously injured patients to life and livelihood, it will have been justified.

THE SURGICAL TREATMENT OF PATIENTS WITH CRANIOCEREBRAL INJURY

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WHEN caring for a patient with craniocerebral injury a capital question which the physician must decide is whether surgical intervention is indicated. The factors so frequently creating a dilemma are first that the patient may have two or more co-existing intracranial conditions and second that one condition may be attended by signs which are supposed to be produced by another condition. Some surgeons[23] believe that operative intervention is indicated in not more than 5 to 10 per cent of patients with head injury. Pilcher and Angelucci[22] reported that among 373 patients fifty-three (14.2 per cent) were operated upon. Nineteen of the fifty-three patients died, an operative mortality of 35.8 per cent. They pointed out that among those who died following operation many had such extensive injuries that the operation played little if any part in the fatal outcome. It is Mock's[18] opinion that in a series of proved skull fractures if the physician operates upon less than 6 or 8 per cent he is overlooking operative indications and his mortality rate will be too high. If, on the other hand, the physician is operating upon more than 10 or 12 per cent of his consecutive run of patients with skull fracture, he is also in danger of increasing his mortality rate. In his series of twenty-nine operative patients eight died (27.5 per cent). Meredith[16] reported that among forty-two patients with craniocerebral injury who were operated upon ten died (23.8 per cent).

To propose an operation conscientiously upon a patient with head injury the surgeon must have in mind some purpose for which his operation is designed. The following are the most common surgical objectives in these cases: (1) Removal of intracranial hematoma (extradural, subdural, intracerebral); (2) removal of perforating foreign body; (3) repair of cerebrospinal fluid fistula; (4) exploration by trepanation; (5) reduction of increased intracranial pressure by decompression; (6) elevation of simple depressed fracture; (7)

débridement of compound wound, and (8) operation against complications (brain abscess, osteomyelitis of the skull).

The purpose of this paper is to discuss each of these indications for operation and to report results in a series of fifty-seven consecutive operative patients with craniocerebral injury who came under my care. Since the paper deals primarily with acute injuries, no patient who had been injured more than six weeks prior to operation was included in the series.

REMOVAL OF INTRACRANIAL HEMATOMA

Removal of an extradural hematoma is usually a neurosurgical emergency. The classical signs of a free interval of consciousness, dilated pupil on the homolateral side, gradual development of hemiplegia and Jacksonian convulsions on the contralateral side are likely to be absent because damage to the underlying brain at the time of the accident may cloud the picture. Van Wagenen[25] and Gurdjian[10] both believe a lucid interval is the exception rather than the rule. It is particularly important to discover the presence of an extradural hematoma since lumbar puncture and dehydration therapy while indicated in cases of edema of the brain are contraindicated in extradural hemorrhage, for shrinkage of the brain in the presence of an extradural clot allows more space into which bleeding may occur. Whenever an extradural hemorrhage is suspected, one or more burr holes should be placed in the skull. It should be kept in mind that extradural hematoma is not always due to middle meningeal artery hemorrhage and therefore is not always found in the temporal region. In three of the four cases in this series the hematoma was encountered through a burr hole in the temporal region. In the fourth, trephining in the temporal region missed the hematoma and it was not until a burr hole was placed high in the parietal area approximately 5 cm. from the midline that the extradural clot was discovered. In two of the four cases the bleeding was from the middle meningeal artery, in one no single large bleeding point could be discovered and in the patient in whom the hematoma was high in the parietal area the source of bleeding was a vein running from the skull into the superior longitudinal sinus. Osteoplastic flaps were turned in three instances and in one the hematoma was removed through an opening rongeured in the temporal region. If the patient is in relatively good condition, I believe the turning of an osteoplastic flap is preferable to rougeuring away the bone because it allows more thorough evacuation of the clot, more adequate control of the bleed-

ing points, and less sacrifice of the cranial vault. In this series none of the four patients operated upon for extradural hematoma died.

The occurrence of subdural hemorrhage is more common than formerly thought. Kennedy and Wortis[15] reported than in their series there were seventy-two cases of acute subdural hematoma while there were only seventeen cases of extradural hemorrhage. They attempted to establish points of differentiation between the two types of hemorrhage. It was their opinion that subdural hemorrhage is likely to occur in all age groups while extradural hemorrhage is more likely to be found in the middle aged and young. In extradural hemorrhage the fracture line might overlie the site of the clot while in acute subdural hemorrhage the lesion more often is contralateral to the skull fracture or site of trauma. Extradural hemorrhage in their opinion is usually unilateral while acute subdural hemorrhage is more likely to be bilateral. In acute extradural hemorrhage, if there is paralysis, it is usually contralateral to the site of trauma while in acute subdural hemorrhage the side of paralysis is less predictable. The lucid interval is likely to be longer in acute subdural hemorrhage than in extradural hemorrhage. Kennedy and Wortis[15] reported that in a series of cases of acute subdural hemorrhage there was a mortality of 43.7 per cent after operation while the mortality in cases of extradural hemorrhage was 54.5 per cent.

Smith[23] advocated that operation for epidural hemorrhage be carried out as soon as the diagnosis is made and the period of shock is over, while he believed it is better to delay operation in cases of subdural hemorrhage for five to ten days if intracranial pressure can be controlled by conservative measures.

Among the fifty-seven patients in this series there were seven who were operated upon within six weeks of injury for subdural hematoma. In my experience many more patients with subdural hematoma come to operation when more than six weeks have elapsed from the date of injury than when less than six weeks have elapsed. Among the seven patients, bilateral subdural hematomas were found in one. Postoperatively, there were two deaths among the seven patients. One of the patients who died had a badly lacerated brain as well as an acute subdural hematoma; the other was in very poor condition at the time of operation. In six of the seven an osteoplastic flap was turned while only burr holes with washing out of the hematoma was employed in the treatment of one patient whose condition was desperately poor.

Gross intracerebral hemorrhages are not common in patients with head injury. Gurdjian[10] reported that in their series of about 400

operations for head injury there were only three instances of massive intracerebral hematoma. Courville and Blomquist[5] reported that among 439 fatal cases of head injury examined at necropsy thirty-six showed gross hemorrhages (in size not smaller than a cherry) within the brain. In my series there was one patient who not only had subdural and extradural hemorrhages but subcortical and intraventricular hemorrhage. Operation in this case was unsuccessful.

REMOVAL OF PERFORATING FOREIGN BODIES

Munro[19] has found perforating wounds of the skull either with or without injury to the meninges and brain are very rare in civilian practice. Any sharp instrument such as a knife or scissors blade, an ice pick or file may produce such a wound. Unless the instrument severs one of the large dural sinuses or a large artery so that severe hemorrhage occurs or penetrates deeply enough to injure vital structures at the base of the brain the outlook for recovery is good. There is, of course, always the possibility that infection will develop. The one patient in my series was a child who fell downstairs and drove the blade of a pair of scissors into her left frontal lobe. The blade broke off flush with the skull leaving a very small skin laceration. The parents, although they could not find the missing blade, had no idea it had penetrated the skull until a roentgenogram revealed its presence. Removal of the blade was followed by an uneventful convalescence.

REPAIR OF CEREBROSPINAL FLUID FISTULA

Meredith[16] believes that when there is an active cerebrospinal fluid leak from the nose, particularly if there is an intracranial aerocele, immediate operation is required as soon as the patient's condition will permit, the communication between the paranasal sinuses and the subarachnoid space being repaired with a fascial strip. Gurdjian[11] stated that when there is cerebrospinal fluid draining from the nose the possibility of intracranial infection is great; but that if the skull roentgenograms do not show fracture lines, particularly fragmentation at the base of the skull, conservative treatment can be undertaken. If conservative treatment is decided upon, the patient should be placed in semi-Fowler's position for four to six weeks; he should be as quiet as possible and never blow his nose. An antiseptic vasoconstrictor drug should be instilled in the nose three times daily. It was Gurdjian's[11] opinion that usually the drainage stopped within forty-eight hours. If the drainage persists for six to eight days or recurs after apparent cure, operative treat-

ment is indicated. I have had occasion to repair chronic but no acute cerebrospinal fluid fistulas. In my experience with six acute cases of cerebrospinal fluid rhinorrhea the drainage always stopped within a few hours after injury and under chemo-therapy infection from this source has not occurred.

EXPLORATION BY TREPANATION

There are patients who have sustained craniocerebral injury in whom the surgeon suspects that an intracranial hematoma may be present even though the classical signs which are supposed to be produced by these lesions are not present. If the patient has been given adequate conservative treatment and fails to show signs of improvement a burr hole exploration of the brain is indicated. In many of the patients the surgeon will find only cerebral contusion, laceration, edema and excessive subdural or subarachnoid fluid. However, an intracranial hematoma will be discovered sufficiently often to justify the frequent performance of this procedure. Some surgeons[11] advocate one trephine opening on each side of the head if subdural hemorrhage is suspected; if the hematoma is not found, encephalography rather than more openings in the skull is indicated. Burr holes can be so easily and quickly made in the skull I generally place three or four on each side. The patient's head is supported in the cerebellar region so that sterile draping of both sides of the head can be obtained. If a hematoma is found on one side, that side is turned up and the remainder of the operation carried out.

In the series being reported twenty-nine patients had burr hole exploration. Included among these twenty-nine patients are those also classified under "Removal of intracranial hematoma" and "Reduction of increased intracranial pressure by decompression" because operations on patients in both these latter groups were started as burr hole explorations and then converted into some other kind of operation the type depending upon what the burr hole exploration revealed. In twelve of the twenty-nine patients intracranial hematoma was found and the operation then directed toward its removal. In six patients burr hole exploration was followed by an operation intended to relieve increased intracranial pressure and in the remaining eleven patients the operation terminated with burr hole exploration only. Among these eleven patients two were found to have a large amount of subdural fluid. Seven had excessive subarachnoid fluid and in two there was no increase in either the subarachnoid or subdural fluid. Of the eleven patients who had burr

hole exploration of the brain which was not followed by any other type of operation four died and seven recovered. The mortality rate in this group is high because I have made it a rule never to let a patient expire without trephining the skull unless he has a head injury of such severity that his case is obviously hopeless. The occasional discovery of an intracranial hematoma in a patient who is practically moribund and who subsequently recovers after operation has made this effort worth while. Two of the patients who had a marked increase of subarachnoid fluid showed very rapid improvement following its release. It is my opinion that the release of subarachnoid as well as subdural fluid in some of these cases justifies the operation even though no intracranial hematoma is found. It has been noted that in some of these cases there was marked edema of the scalp following trephining, indicating that an excessive amount of fluid was draining through the burr holes.

REDUCTION OF INCREASED INTRACRANIAL PRESSURE BY DECOMPRESSION

The majority of surgeons handling patients with head injury now believe that operations devised primarily for decompression of the brain are not advisable. While the decompression will increase the size of the intracranial cavity, frequently the swollen brain herniates through the opening causing tears in the brain and hemorrhages. In six of the twenty-nine patients who had burr hole exploration this procedure was followed by an attempt to reduce the intracranial pressure through decompressive measures. In all these cases it had become obvious that conservative methods were destined to failure; and even though it was known that "the dice of the gods were loaded," decompression was performed. Two of the patients had unilateral subtemporal decompression, accomplished by enlarging a burr hole with the rongeur; three had bilateral subtemporal decompression done in the same manner. One had a large temporoparietal block of bone removed by means of the burr and Gigli saw. Operation in none of these six cases saved the patient's life. This bears out the opinion that decompressive operations of this sort are of little or no value.

Briesen and Jones[2] suggested that increased intracranial pressure following head injury can be relieved by placing a drain in the cisterna magna and they reported one successful case. Ody[20] reported a similar operation. It has been noted on numerous occasions that a patient who drains cerebrospinal fluid from his ear or nose, if he does

not develop infection, recovers more rapidly than the one who does not have a loss of cerebrospinal fluid. This might indicate that the suggested operations of Briesen and Jones[2] and Ody[20] are of value.

ELEVATION OF SIMPLE DEPRESSED SKULL FRACTURES

Patients with simple depressed skull fractures should be operated upon for cosmetic purposes and to relieve pressure upon the brain. In most instances the elevation of the depressed fragments is not a surgical emergency although it should be carried out before the fragments become too firmly fixed in malposition. Walker[27] stated that the indications for early operation are continued convulsions which could not be relieved by dehydration and decline in the patient's condition in the absence of some other cause. Glaser and Shafer[8] concluded that early elevation of the depression did not prevent late sequelas. It seems that epilepsy is more likely to be due to the formation of a cerebral cicatrix than the irritating effects of a depressed bone fragment. Among six patients in this series operated upon for the elevation of simple depressed fracture there were no deaths.

DÉBRIDEMENT OF COMPOUND WOUNDS

A high proportion of patients suffering head trauma will sustain compound wounds. This is particularly true among war casualties. Although the maxim "treat shock first" is continually ringing in one's ears, only a small percentage of patients with craniocerebral injuries are in shock on arrival at the hospital. Meredith[16] stated that less than 10 per cent of any large series of patients with head injury of all types are in shock.

Certainly as soon as the patient's condition will permit, a débridement of the wound should be performed provided adequate facilities are at hand. Grant[9] has pointed out that two lessons seem to stand out from the first World War: (1) Patients with cranial trauma can be transported with greater safety before operation than immediately afterward; (2) when the patients with craniocerebral injury are operated upon by specially trained men in properly equipped centers they do better than when they are assigned to the general surgeon. Jentzer[14] remarked upon the importance of having complete neurosurgical equipment and thought that the necessary quiet for a neurosurgical operation could not be found closer than twenty-five kilometers behind the battle line. He thought that the patients following operation should not be transported for at least fourteen

days. Tönnis,[24] in speaking of the treatment of patients with brain injury sustained during the campaign against Poland, stated that operations attempted in unsuitable conditions made the patients worse. Those who had the good fortune to be transported to a base hospital were the only ones who did well, and those who were transported by air did the best. Cairns[4] agreed that if the patient's condition was unfavorable or adequate operating facilities were not at hand it is better to delay operation. He stated that brain tissue resists and localizes infection as well if not better than other tissues.

Horrax[12] suggested that three prophylactic measures should be instituted in the treatment of patients with war wounds of the brain: (1) Soldiers should go into action with closely cropped hair; (2) tetanus toxoid should be given to all soldiers and civilians likely to be involved in enemy actions; (3) the sulfonamides should be given by mouth as soon as possible after any kind of compound injury has been received.

When a patient with a compound skull fracture is first seen, the hair about the wound should be clipped or shaved, one of the sulfonamide drugs sprinkled in the wound and bleeding from the wound controlled by packing with gauze. These procedures are particularly important if for any reason some hours are likely to elapse before a careful débridement can be done.

The best anesthetic for the débridement is novocain although if the patient is exceptionally violent avertin or pentathol sodium may be necessary. The patient can be spared some pain if the surgeon will shave and clean the scalp at a considerable distance from the wound. Novocain can then be infiltrated into the scalp around the wound at some distance from the edges. Following this, cleansing of the wound edges with tincture of green soap, ether, alcohol, and tincture of merthiolate will be painless. When the region is prepared, one should drape a large area because it may be necessary to extend the skin incision for some distance to expose adequately the defect in the bone. Ragged skin edges are excised and the knife discarded. Hair and débris are removed from the wound and copious quantities of warm saline solution employed for washing the wound. The extension of the skin incision, in order to secure adequate exposure, may be done in several ways. If there is loss of scalp, the tripod incision or the three-legged (Isle of Mann) incision may be employed to facilitate co-aptation of the wound edges. Where the scalp wound is small, a horseshoe-shaped skin flap with the wound of entry in the middle may be turned as suggested by Winslow.[28]

In order to elevate the depressed bone fragment it may be necessary to place a burr hole adjacent to the edge of fracture and cut along the fracture line with the deVilbiss. The depressed bone is lifted out in as large pieces as possible; these are taken by the surgical nurse and thoroughly cleansed with saline solution. When the depressed bone fragment is lifted out there may be sharp bleeding if one of the venous sinuses has been torn by the bone edge. This bleeding can be controlled with a muscle stamp. The removal of all cracks in the bone in which infection may track has been suggested.[25] It seems to me that this would be impractical unless there is gross débris in the fracture line since in many patients linear fractures radiate out for several inches from the area of depression. While most surgeons nibble away the bone edges to remove the depressed fragment and gain adequate exposure Burckhardt,[3] Garcin and Guillaume,[7] and Vincent and Lafon[26] turned osteoplastic flaps to expose the penetrating wounds. Ducuing[6] found that in most cases fractures in the skull were so extensive that the flap broke to pieces when this method was employed.

If the dura is intact and there is no evidence of hemorrhage underneath, as indicated by a tight dura or discoloration, it should not be opened. When the dura is lacerated and the brain substance damaged, the necrotic brain tissue is removed with the suction apparatus, care being taken not to encroach any more than necessary upon nerve pathways which are still intact. Also one should be careful not to open into a ventricle or basal cistern since this increases the danger of infection. Bullet and bone fragments are removed under direct vision if they are accessible. A rubber catheter is excellent for exploring the track in the brain. By means of the sucker the track is converted into a smooth-walled cavity. If the bullet has traversed the brain to an inaccessible position, an attempt should not be made to remove it. Ascroft[1] analyzed 317 cases of gunshot wounds of the head (patients with wounds of the cerebellum were excluded from this series) sustained during the 1914 to 1918 war. He found that 34 per cent developed epilepsy but that there was no evidence that removal of metallic foreign bodies from the brain diminished the liability of this complication. He thought prolonged wound sepsis favored the subsequent development of epilepsy both in those cases in which the dura had been penetrated and in those in which the dura had remained intact.

The dura should be closed if possible. When there is a communication between the subarachnoid space and one of the paranasal

sinuses, it is imperative that a tight closure be affected, taking if necessary fascia lata, fascia from the temporal muscle or periosteum from under a scalp flap to affect the plastic closure. If the compound fracture is over the vault and the dura cannot be adequately closed, a small opening in the dura is not of much importance if a good skin closure can be affected. In these cases the opening in the dura can be covered by a nonadhesive membrane manufactured for such a purpose.

In civilian practice the pieces of the skull which have been thoroughly washed with saline solution can be replaced and a firm closure obtained. It may be necessary to hold the fragments together with sutures passed through drill holes in the pieces of bone. The skin is closed with interrupted No. 00 plain catgut to the galea and interrupted fine silkworm sutures to the skin. If the laceration is in the forehead, a fine Kal-dermic (6-0) or horsehair suture is used in the skin. No drains are left in place unless there is evidence of infection at the time the débridement is performed.

The local use of the sulfonamides in the wound has come in for much discussion in the last few years. Mitchell, Logie and Handley[17] in commenting on experiences in the British campaign in Libya stated that the use of these drugs cannot take the place of careful treatment of war wounds. It is still a debatable question which of the drugs is most effective locally in the brain and which is least likely to produce an untoward effect. Experimental work[21] which may help to solve this problem is now in progress. Hurteau[13] found that after local application to cerebral wounds the several drugs of the sulfonamide group show a difference in the rate of absorption. According to him sulfanilamide is the most rapidly absorbed, sulfathiazole the second, sulfadiazine third, and sulfapyridine the least rapidly. He suggested the mixture of several of the drugs, for example sulfanilamide and sulfadiazine. A high concentration of sulfanilamide would occur immediately while a high concentration of sulfadiazine would persist for a time to cope with latent infection.

In my series of nineteen patients who sustained compound skull fracture thirteen had laceration of the dura and in seven of the nineteen there was loss of brain substance. The time which elapsed from injury to operation ranged from three to thirty-four hours, the average elapsed time being ten and one-half hours. While one likes to operate upon these patients within six hours of the time of injury, many patients in this series were sent in from some distance so the average time from injury to operation was well beyond six hours.

Thorough débridement was carried out in each case and in fourteen patients the pieces of bone were replaced so no defect would remain in the skull after healing. In fifteen the skin edges were closed tightly while in three a small Penrose drain and in one a small wick of gauze was placed underneath the skin. One patient with an extensive laceration of the brain died seventy-two hours after surgery and the other eighteen recovered. All wounds in the eighteen patients who recovered healed without infection and there was no evidence of infection in the wound of the patient who died. In two instances there was a slight amount of cerebrospinal fluid drainage for a few days. Sulfanilamide or sulfathiazole was sprinkled in the wounds of seven patients but it is difficult to ascertain the efficacy of the sulfonamide drugs applied locally in these cases since infection did not occur in any of the patients.

OPERATIONS DIRECTED AGAINST COMPLICATIONS

A complication such as brain abscess, osteomyelitis of the skull, or meningitis may follow craniocerebral injury. Usually the osteomyelitis is not the rapidly progressive fulminating type which frequently occurs secondary to infection in the paranasal sinuses. It cannot be overemphasized that the best treatment for osteomyelitis of the skull and brain abscess is preventive. No craniocerebral injury or even simple scalp laceration should be neglected. All should be thoroughly cleansed, repaired and bandaged.

In this series one patient who sustained a fracture through the base of the skull with the subsequent development of osteomyelitis of the cribriform plate and abscess underneath the frontal lobes was operated upon but did not survive. Another patient had a scalp laceration sutured by his local doctor who did not realize a compound skull fracture was present. Subsequently a subdural abscess formed. Following removal of the bone fragments, drainage of the abscess and vigorous chemotherapy he finally recovered.

CONCLUSIONS

1. Twelve patients with intracranial hematoma were operated upon and nine of the twelve recovered (mortality 25 per cent).

2. Patients who do not improve after adequate conservative treatment should have burr hole exploration of the brain for there is no other way to exclude conclusively the possibility of an intracranial hematoma.

3. Removal of portions of the skull for the primary purpose of reducing intracranial pressure did not prove effective.

4. Careful débridement of compound craniocerebral wounds will be attended by a high percentage of recoveries. In this series eighteen out of nineteen patients with compound wounds recovered (mortality 5.3 per cent).

5. The mortality rate for fifty-seven patients with craniocerebral injury operated upon within six weeks of injury was 26.3 per cent.

TABLE 1

OPERATIONS UPON FIFTY-SEVEN PATIENTS WITH ACUTE CRANIOCEREBRAL INJURY*

Purpose of Operation	No. of Patients	Deaths
Removal of intracranial hematoma		
Extradural	4	0
Subdural	7	2
Intracerebral	1	1
Removal of perforating foreign body	1	0
Exploration (burr holes)	11	4
Reduction of increased intracranial pressure (decompression)	6	6
Elevation of simple depressed fracture	6	0
Débridement of compound wound	19	1
Against complications		
Brain abscess	1	1
Subdural abscess	1	0
Total	57	15 (26.3 per cent)

* All of the patients in this series were operated upon within six weeks of the time of injury.

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DISCUSSIONS OF PAPERS OF DR. KING AND DR. RAAF

DONALD MUNRO (Boston, Mass.): I have been privileged to receive copies of Dr. King's and Dr. Raaf's papers, and I am sorry that time did not permit the authors to read them in detail, because you would have been well repaid by having heard them read in that way. I recommend that all of you read them in detail when they are published.

The contents of these papers do not allow for any discussion. Everything that is said is without doubt true, and I am in complete agreement with the subject matter in both papers, except for certain minor details.

Because of the impossibility of discussing an undiscussable paper, it seemed to me that I might avail myself of this opportunity to present to you certain unpublished data in regard to some of the high spots of these problems of intracranial hematomas and compound fractures of the skull.

I should like to speak first about extradural hematomas. I do not look upon extradural hematomas with the same equanimity as Dr. King and Dr. Raaf do. In my hands, I am of the opinion that extradural hematomas presented one of the most difficult problems of diagnosis that the traumatic neurosurgeon has to face. The classical syndrome by which the diagnosis is said to be possible has, in my hands, been so rare as to be cause for calling the house officers and other members of the staff around, so they could see an example of this curious, interesting phenomenon.

In a group of thirty-seven extradural hematomas, in ten cases the clot resulted from venous hemorrhage. I should like to emphasize that, because I think that is not very well recognized, and I think that it is important because it is in these cases, in particular, that the patients live a relatively long time between the infliction of the wound and their death.

In regard to the diagnosis, in my group of cases only 40 per cent showed a lucid interval. That is considerably less than one would be led to expect from reading the ordinary surgical textbooks; and I commend to you the fact that you will probably see few extradural hematomas that show the pathognomonic triad with the lucid interval between two periods of unconsciousness, and you will see patients with extradural hematomas who remain constantly unconscious from the time they received the blow until you operate upon them.

In support of that, 50 per cent of my patients were unconscious on admission, and 92 per cent had bloody cerebrospinal fluid, which led us to conclude that all of those patients had at least a contusion of the brain with the associated uncounsciousness that goes with contusion of the brain.

In regard to subdural hematomas, these are a very common complication of craniocerebral injuries. They are, in the acute cases, in my experience, diagnosable only by exploratory trephination, and in connection with that, I think that it is quite possible that as we get more experience and as you general surgeons begin to take up part of this load, you will find, and we will find, that multiple exploratory burr holes will be more and more common. By multiple exploratory burr holes I mean not two burr holes, one on either side, but I mean many burr holes done in all areas of the skull until the hematoma is actually demonstrated or until an adequate spotty exploration of the contents of the skull has been carried out.

In connection with that, it has recently been recommended that this exploration be carried out with the patient in the cerebellar position so that

burr holes can be placed over the posterior fossa as well as over the two lateral fossae, and in that way the cerebellar subdural or extradural hematoma be identified.

In my experience, I have had 310 patients with subdural hematomas. Practically all of them have been operated upon, and in all of them the subdural hematoma has been the major complicating factor. One hundred ninety-four of these 310 cases were associated with major brain injuries, and I believe that this type of subdural hematoma, the subdural hematoma that is caused by the associated brain injury, is far and away the most important variety of subdural hematoma and is the one that yields most readily to the immediate operation done within a short time after the entrance of the patient into the hospital. In this group my mortality has been 40 per cent. These mixed hematomas, if they are not operated upon, go on to an end result of a cystic collection of fluid which is free from the subdural space. That has been described formerly as a hydroma. I prefer to consider it a hematoma, because they all start out as a mixture of blood and cerebrospinal fluid, and because a hydroma requires by definition that the fluid be contained within a sac. Hydroma as a name could be more properly applied to the end result of the solid subdural hematomas.

There were seventy-one of these. Of the solid clots, there were only forty-five. These are the clots that were described by Cushing and Putnam.

Just a word about compound fractures. The greatest problem, probably, in compound fractures today is when to operate upon the compound fracture and what kind of first dressing to apply. The latter is particularly true because of the present use of the sulfonamide products. In an endeavor to find out when to operate upon compound fracture wounds, I have recently gotten together statistics from fifty-six cultures made on fifty-six compound fracture wounds. It was interesting to note that contamination, although doubtless present in every wound, was culturable in only 55 per cent during the first twenty-four hours, and in only 66 per cent during the first forty-eight hours, whereas after forty-eight hours, 91 per cent of these wounds showed easily culturable bacterial contamination. It seems to me in view of this, and in view of my experience, there is never any hurry about operating upon compound fractures of the skull, provided you do the operation within the first forty-eight hours of the receipt of the wound, and provided, and this is equally important, you do not manipulate the wound at the time of the first dressing.

In connection with that, I would like to say that the results published by Cloward, in Pearl Harbor, it seems to me, are dangerous, because he suggests that at the dressing station the patients with compound fracture wounds not only had their hair clipped but that the scalp was shaved and the wound was scrubbed at that time. This I consider to be a very dangerous procedure because I am sure under that amount of manipulation there will be a wider spread of contamination into the wound than would otherwise exist if the wound were left alone until the major débridement is done.

In regard to the use of sulfonamide products in these wounds, there is no question but that is a very valuable adjunct to our surgical procedure in dealing with compound fractures. However, do not use sulfathiazole on the surface of the brain. Recently a series of cases have been reported—and there are some others that have not been reported—in which the use of sulfathiazole on the surface of the brain, both experimentally and in the human being, has produced epileptiform seizures which were traceable directly to the use of that particular drug. If you want to use the sulfonamides in the wounds themselves, use sulfanilamide or sulfadiazine powder.

Of course, the sulfonamides should be given by mouth from the first time that you see the patient.

In closing, I would like to congratulate this society and congratulate the two readers on having brought this important general surgical problem to the notice of general surgeons, and taken it away from the narrow specialty, where it has been resting for the most part, to the present. These problems of craniocerebral surgery are general surgical problems. More and more of the general surgeons are going to have to face them and deal with them, and it is well that that should be brought to the attention of a general surgical society such as this.

WILLIAM JASON MIXTER (Boston, Mass.): Dr. Munro said he was not going to discuss these papers. I think he did a pretty good job of it, and I do not think he left me much to discuss.

They are good papers, and they are papers of great interest to a group of men who are handling the surgery of trauma, not only for the reasons that Dr. Munro has given, but also due to the fact that even if you do not want to handle these cases yourself, sometimes you are forced to, just the same as Dr. Munro or Dr. King or I do when we go to some little hospital out in the sticks around Massachusetts to see a patient with a severe head injury, and there we find a tibia scratching around in the sheets and nobody has done anything to take care of it before we got there. That happened to me within the last two years, and the compound fracture of the tibia was a more important feature than the head injury.

The war brings up the same problem. Even if we wanted to put a neurosurgeon in every front-line hospital, it could not be done. There are not enough of them. The result is that the general surgeons must pick up a considerable group of these cases and handle them, and handle them intelligently. For that reason, I commend these papers to you, as Dr. Munro already has.

JOHN S. HODGSON (Boston, Mass.): I have found Dr. King's and Dr. Raaf's papers of great practical interest. Traumatic hematomas, whether they are extradural or subdural, or the rare subcortical ones, plus compound and depressed skull fractures and complicating spinal fluid leakage from cranial wounds and orifices, intracranial aerocele and infection of the scalp, bone, meningeal spaces and brain itself, represent the conditions calling for

cranial or intracranial surgery. It seems to me that the general surgeon can be of definite help in traumatic neurosurgery in these times.

The papers have been so well given and completely discussed that it is difficult to know just how much to say. I am going to limit my remarks except to say that the traumatic extradural hemorrhages are rather rare, comprising a very small percentage of the cases of head injury, but they are so serious that their rarity makes them all the more important. Whether or not one uses a burr hole and enlarges it, or uses a bone flap, is somewhat unimportant, probably. I think that most of us probably simply remove enough bone to get at the bleeding point, which is usually under the temporal muscle, but if there is some question as to the location of it, it may be better to make a bone flap.

While he did not speak of it, Dr. King wrote in his paper about sometimes needing to tie off the external carotid artery in cases in which the extradural hemorrhage was not easy to locate, or in cases in which it was extreme in degree and the patient's condition might be such that time was very valuable. I think in such a case it might be all right to tie off the external carotid artery, but that must be a rather rare necessity, of course.

I think anyone doing the surgery of extradural hemorrhage must be prepared to follow through to the point where the hemorrhage is coming from, even if that involves going to the base of the brain, as far as the foramen spinosum, and plugging that foramen with cotton. I think it is better to do that than to do the ligation of the external carotid, although the latter may be necessary in rare instances.

In regard to the subdural hematomas, those, of course, are much more common and so much has been said abo[illegible] those that I am not going to attempt to add anything at this time.

The subcortical hematomas are extremely rare, and yet they do occur, and when one has decided to operate and does operate, and does not find what he thinks he is going to find, namely, a subdural hematoma, he should be prepared to look at least locally for a possible subcortical hematoma. That was brought home forcibly to me again recently when I operated upon an elderly person whom I suspected of having a subdural hematoma, which he did not have. He had no localized signs at all but the cortex appeared a little abnormal in that region, and on tapping it, I found a rather deep and large subcortical hematoma. Therefore, one should be eager enough to follow through, to look for a possible subcortical hematoma. It will be rare, but it occurs just often enough to necessitate one's always keeping it in mind and looking for it every now and then. Of course, it may occur along with extradural or subdural hematoma as well.

Regarding the repair of dural rents in cases in which there may be spinal fluid leakage from a head orifice, I should say that that is a pretty major operation, and it would seem as though the general surgeon would do well not to take on a case of that kind, deliberately, at least, because it does

involve pretty complete experience with neurosurgery. It means making a large scalp incision, turning back a bone flap, looking deep in the cranial fossa, finding the rent in the dura and then sewing it up. It is not easy to sew up rents in the deep dura. You are told that all you need to do is sew the edges together, but they are so thin and friable that they cannot be sewn together easily, so that you either have to use a piece of fascia, which often does not work well, or a piece of muscle. I have found that a piece of muscle is rather good to put in between the edges of the dura that will not come quite together. If one will stitch the edges together with a piece of muscle in between, he will get a good closure which he otherwise could not get at all.

Also, it is a good plan sometimes to take a piece of muscle and cover over places like the cribriform or some other place where there is a fracture in the base of the skull. That will do what nothing else can do.

I had an interesting case recently which I think it will be worth while mentioning. It was a child of eleven who came into the Massachusetts General Hospital with the history of having had a head injury at the age of four, seven years previously. There had been bleeding from the ear which had stopped, and the child had apparently been all right, as far as we could find out from the family, during the succeeding seven years. Just before coming in, she developed a spontaneous leakage of spinal fluid from the ear on the side that had leaked seven years before. She had come into the ear and nose clinic and was transferred to the neurosurgical service. *X*-ray showed a fracture in a portion of the temporal bone. We operated, took out a large piece of bone in the temporal region, made drill holes and lifted out the dura and found the rent in the dura. We could see the fracture line in the bone still. The dura had funneled into the fracture line in the base of the skull. The dura was pushed back, the fracture line in the skull was sealed off with bone wax, and the dura edges were brought together with a little difficulty because they were thin and friable and I had to put a piece of muscle in between and close it in that fashion. Healing was complete and she got along very well.

That to me was a rather unusual case. I have never personally happened to have to repair a cerebrospinal fluid orrhorrhea before. They all leak, if they leak at all, for a few days and then stop. The repair was done in the same fashion that you would repair a cerebrospinal rhinorrhea.

One other case that interested me a good deal in regard to a repair was that of an elderly man who had had a severe head injury. He had been kicked by a horse. It had made a straight up and down scalp wound. He had been seen by a local physician who took charge of him and did the original surgery. At the time of the injury, this horse that kicked him kicked out a large piece of skull in the left temporoparietal region. At the time the man was picked up, the piece of bone was seen in the street and picked up along with the man and taken to the hospital. The scalp wound was sutured;

the dura was not lacerated much and he had not suffered much intracranial damage. The doctor simply sewed up the scalp wound and kept the piece of bone in saline.

When I saw the man a few weeks after, the wound was not quite healed, and we waited three weeks more. He was transferred to the Massachusetts General Hospital and the piece of bone came along with him. That was sterilized, the wound was reopened and the piece of bone put in. That operation was finally done about eight weeks after injury. It did not completely fill in the space because a little had been broken out in small pieces in the low temporal region, but that was put back in and sutured in with small drill holes and suture material, and the wound healed well and has remained healed since that time, a matter now of several months.

JOSEPH E. J. KING (closing): I necessarily had to read my paper in such a sketchy manner that I did not cover all the points, and I believe if Dr. Raaf will wait until he reads my paper, he will find he agrees with me, and I certainly agree with him except about the bone flap. You must remember that these people are sick and you cannot fool around and turn up nice bone flaps. What you want to do is to get the blood clot out and get the patient back in bed, and I can do it through one or two holes much quicker than by making a bone flap. If you make a bone flap you have to make a lot of holes, too.

Most blood clots come from the middle meningeal injuries.

It is true that ligation of the external carotid will very seldom be used, but you should not send a patient back to bed when you cannot see where the blood is coming from. You know how it comes out like a waterfall sometimes. Pack it and make sure the packing is secure, but rather than risk it, I would ligate the carotid. I had to do that for a patient with unilateral nose bleed. He would have died otherwise. He had had five transfusions and we could not stop the hemorrhages, and I finally had to ligate.

Dr. Munro said something about Dr. Raaf and myself viewing these cases with equanimity. No, we do not do that. I know they are serious cases and I look at them very seriously.

In the case of venous hemorrhage, of course, especially in the posterior fossa, I agree with Dr. Munro that you must have exploratory trephination all over, not as many openings as in a chair back, but several openings are much better than to send a fellow back and not find it. I even put in the paper what a humiliating feeling it is to operate upon some patient for a clot which you do not find, and when he comes to autopsy examination there is a big one you missed.

JOHN E. RAAF (closing): On this matter of the bone flap, I will agree with Dr. King that if you stand by and watch the patient with an extradural hematoma until he is in a serious condition, the best method of treatment is to put in a burr hole, enlarge the burr hole and suck out the clot. However, if the patient is in good condition, I would rather turn a bone flap,

because even though a single bleeding point may start the hemorrhage, as the hematoma develops and strips the dura away from the bone, numerous bleeding points will start. There are blood vessels running between the dura and the bone which will start to bleed as the clot develops, and you can more adequately control these if you have a large flap.

I will agree with Dr. King that I did not mean to create in Dr. Munro's mind the impression that I thought the diagnosis in extradural hemorrhage is easy. It is difficult, and I think that is the reason many of us are coming to the point where we put multiple burr holes in the skull. It has been advocated lately by at least one surgeon that one burr hole should be placed on each side and if the hemorrhage is not found, one should resort to other studies, such as encephalography. I disagree. I think multiple burr holes are indicated, and sometimes I operate upon patients in a sitting position so that numerous burr holes can be placed in the skull, including the occipital area.

TRAUMATIC RUPTURE OF THE SPLEEN*

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TRAUMATIC rupture of the normal spleen is not the clinical rarity it was formerly thought to be. Its increased incidence is both real and apparent. On the one hand, this is due to better diagnosis; on the other it is accounted for by the year-to-year increase in automobile and industrial accidents. It is also important to note that in reports thus far available from England ruptured spleen is high on the list of abdominal injuries incident to blast.

The exact incidence of traumatic rupture of the spleen is difficult to determine. That it is higher than is commonly recognized is one of our fundamental conclusions. That the diagnosis is too frequently missed we know from experience at our own institution. This is one of the prime reasons for again calling the attention of the profession to this result of trauma. Working as we do in a general hospital which serves private patients as well as a teaching institution for the medical school, we have the opportunity to observe the methods of a considerable group of surgeons in the management of accident cases. During the present year, two cases (Nos. 19 and 20) of traumatic rupture of the spleen remained undiagnosed until necropsy.

In reviewing the pertinent literature one is impressed by the large number of papers reporting one or two cases of splenic rupture, indicating that many surgeons still consider it rare enough to warrant a paper composed of a single case report. That the true incidence is far greater is indicated by the report of Wright and Prigot.[48] They found traumatic rupture of the spleen in one out of every 666 accident cases of all kinds admitted to the Harlem Hospital in New York City.

At the University Hospital our incidence has been computed to be one out of every 920 accident admissions of all types.

* From the Department of Research Surgery, The Ohio State University. This investigation was aided by a grant from the Comly Fund of The Ohio State University.

This report is based on a series of twenty-two cases of traumatic rupture of the spleen occurring in the city of Columbus, Ohio, during the years 1929 to 1941 inclusive. This period has been chosen because it begins with the adoption of uniform record library methods throughout the city. The cases reported are all those recorded in the files of the city's six hospitals during the period. Our own cases total eight in number and represent one-third of the group. We believe that this group represents an accurate picture of the occurrence, recognition and management in a fairly typical mid-western city of 300,000 inhabitants. It affords a good basis from which to appraise both the mistakes and successes in the present day surgical management of traumatic rupture of the spleen.

ANATOMY AND PHYSIOLOGY

A brief review of the anatomy and physiology of the spleen is of considerable help in a better understanding of the pathology and treatment. The spleen is a vascular, lymphoid organ measuring normally about 12 by 7 by 4 cm. and weighing about 200 Gm. Its tissue is soft and friable. Lying as it does between the fundus of the stomach and the diaphragm the spleen is normally protected by the lower portion of the thoracic cage. It is almost entirely surrounded by peritoneum and is held suspended by two folds of this membrane, the phrenicolienal ligament and the gastrolienal ligament. The friable, fragile nature of the splenic tissue, together with its mode of suspension explain the frequency with which the spleen is ruptured by indirect force despite its seemingly protected position. Also, since the large vessels supplying the spleen run through the gastrolienal ligament, it is easily seen that a tear of this ligament would result in grave hemorrhage.

The splenic artery, next to the renal arteries, is the largest vessel supplying any of the abdominal viscera. It is the largest branch of the celiac axis, and is out of all proportion to the size of the organ it supplies. The artery divides into a series of branching networks which finally allow the blood to percolate through the interstices of the reticulo-endothelial tissue. Thus it may be seen that, for the purposes of our consideration, the spleen may be thought of as an organ made up largely of blood vessels and spaces for blood within the loose pulp. A fracture in its substance may be considered as a tear in one of the most vascular tissues in the body.

The nerve supply to the spleen comes from the celiac plexus. Stimulation of these autonomic fibers leads to contraction of the smooth muscle fibers within the capsule and trabeculae.

The physiology of the spleen is still not completely understood. However, three well recognized functions may be stated, namely, the destruction of formed blood elements, the storage of blood, and the production of lymphocytes in the Malpighian corpuscles. It is with the second of these functions that we are particularly concerned in this discussion.

Stuckey, as early as 1742, stated that the spleen acted as a blood reservoir. But it was not until the work of Barcroft[3] was published (1922 through 1936) that the dynamics of this function were understood. Through these studies as well as the work of others, Holman,[24] Hargis and Mann,[32] it is now known that the spleen contracts in asphyxia, exercise, hemorrhage and emotional excitement. Doan, Curtis and Wiseman[16] actually demonstrated the contraction of the human spleen by injecting adrenalin into the splenic artery at operation and noting its diminution in size. In effect, this actually results in a largely cellular autotransfusion. This procedure together with massage is used routinely by one of us (G. M. C.) in the course of splenectomies for pathologic spleens.

Still more important was the demonstration by Barcroft and Nisimaru[3] that the spleen undergoes constant, spontaneous, rhythmic contractions. These have a duration of from twenty-five to fifty seconds and are reflected as low undulations in blood pressure tracings. The practical import to our later discussion of treatment is obvious. The surgeon who contents himself with suture or packing and is aware of these contractions, can hardly conclude that he has used the safest method of arresting splenic hemorrhage.

ETIOLOGY

The cause of traumatic rupture of the spleen is some form of direct or indirect physical violence. Less frequently the rupture is due to a penetrating wound.

Many pathological conditions cause enlargement of the spleen. That these spleens are subject to traumatic rupture is revealed in the reports of Banerjee[2] and Henderson.[22] Some authors suggest that the pathological changes which these spleens have undergone make them even more susceptible to traumatism and consequent rupture.

That the normal human spleen may rupture spontaneously has been suggested. Susman[41] collected seven and Zuckerman and

Jacoby[49] collected twenty cases purported to be of this type. However, a more critical review of the available literature casts some doubt on the probability of such an occurrence. It is more likely that these were instances of delayed rupture of previously traumatized spleens; the history of trauma being overlooked or even forgotten. We are hesitant to conclude that spontaneous rupture of a normal spleen can occur.

TABLE I

SEX INCIDENCE OF TRAUMATIC RUPTURE OF SPLEEN

Sex	Incidence
Female	2
Male	20
Total	22

TABLE II

INCIDENCE OF TRAUMATIC RUPTURE OF SPLEEN BY AGE GROUPS

Age Group	Incidence
1–10	5
11–20	7
21–30	4
31–40	2
41–50	2
51–60	1
61–70	1
Total	22

TABLE III

VARIOUS TYPES OF ACCIDENT PRODUCING TRAUMATIC RUPTURE OF THE SPLEEN

Type of Injury	Incidence
Athletic	1
Automobile	11
Bicycle	1
Bullet wound	2
Fall	5
Industrial	2
Total	22

Traumatic rupture of the spleen occurs more frequently in males (Table I); only 9.1 per cent of our patients were women. Likewise age is an important influencing factor. Young individuals are far more often affected than older people (Table II); 81 per cent of our cases occurred before the age of forty years. This may readily be explained by the greater activity of youth.

The manner in which splenic injury is ordinarily sustained is significant. In practically all recent reports, including our own, automobile accidents have accounted for the greatest number. (Table III.) In our series, automobile accidents accounted for eleven cases, or

half of the entire group. It is interesting to glance back at the excellent earlier discussion of the subject by Conners,[13] in 1921, and find that the automobile's predecessor, the horse and wagon, was then the most frequent cause of the injury. A bit of irony is to be found in the report of Dretzka,[18] from Detroit, the capitol of the automobile world, where not the automobile, but gunshot and stab wounds accounted for the largest number of injuries.

Clear-cut industrial accidents account for but 9 per cent of our cases. This suggests that the long campaign for industrial safeguards is having some effect. Falls account for the largest number of the injuries in children.

It is interesting to note that in Gordon-Taylor's discussion[21] of war injuries occurring in England, a new and more bizarre cause of traumatic rupture of the spleen is mentioned. This is rupture due to blast injury. In this respect the spleen is the most frequently affected abdominal organ.

Considering at length the available reports of war injuries, we find that in military as in civilian surgery, the spleen is the most frequently affected organ in subcutaneous injury to the abdominal viscera. The frequent involvement of the spleen in civilian surgery is shown by the reports of Butler and Birnbaum,[11] Bronaugh[8] and Wright and Prigot[48] who find the spleen involved in 30, 33 and 47 per cent, respectively, of all subcutaneous abdominal injuries. Rupture of the spleen is even more frequent in occurrence than rupture of the liver or kidney which follow it in frequency.

PATHOLOGY

From examination of the spleens removed at operation and during necropsy, it is found that traumatically ruptured spleens can usually be classed in one of five categories. These more or less arbitrary groups are illustrated in Figures 1, 2, 3 and 4. Each type corresponds with a different clinical picture. This knowledge is of aid in both diagnosis and treatment.

Type I is a complete fragmentation of the spleen into two or more parts, or a complete tearing of the spleen from its pedicle. This results in immediate massive hemorrhage and in many instances in sudden death. Figure 4 illustrates a spleen of this type, removed in Case 9 of our series. The patient was kicked in the back while playing ball. He rapidly went into shock. He was operated upon within two hours of the injury and made a complete recovery. (Fig. 1.)

Type II represents a large tear at or near the hilus. This gives rise to the rapid onset of grave hemorrhage and like Type I demands the most rapid surgical intervention. (Fig. 2.)

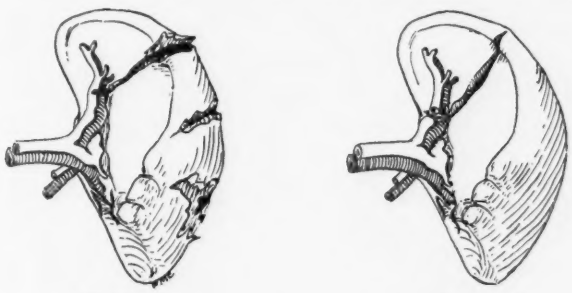

FIG. 1. Diagrammatic representation of Types I and II of splenic rupture, both leading to rapidly progressive hemorrhage. Variations of Type I are a complete severance of the pedicle and a complete disruption of the spleen (Fig. 4).

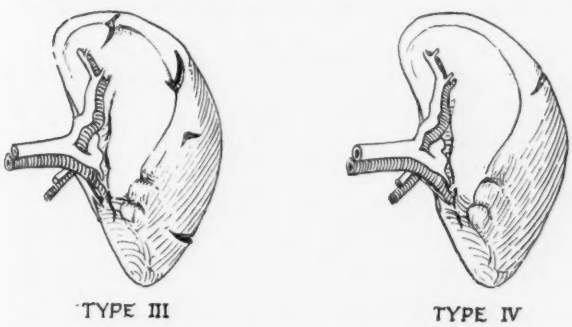

FIG. 2. Diagrammatic representation of Types III and IV of splenic rupture. Type IV is the lesion commonly responsible for the development of a perisplenic hematoma.

Type III is characterized by one large or multiple smaller tears about the periphery of the spleen. This type leads to slow but steady development of the signs of intra-abdominal hemorrhage. (Fig. 2.)

Type IV presents a solitary small tear at the periphery. (Fig. 2.)

Type V is characterized by subcapsular hematoma. (Fig. 3.)

The last two types are those particularly responsible for the development of the syndrome of *delayed hemorrhage* which will be discussed under symptomatology.

One further pathologic consideration is of importance. This is whether or not spontaneous cure of a splenic hematoma is possible. If such is possible, there is some justification for those who believe that this lesion should be treated conservatively. R. C. Webb[45]

quotes J. S. McCartney as reviewing 25,000 necropsies at the University of Minnesota. In this large group he found no instance of a healed splenic hematoma. It is our conclusion that such a spontaneous cure rarely if ever occurs.

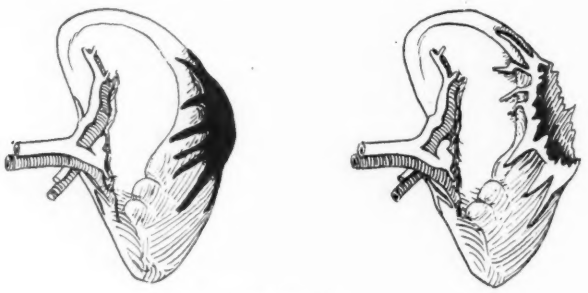

FIG. 3. Diagrammatic representation of Type v splenic rupture. This presents two stages in the evolution of a subcapsular hematoma, the last representing its rupture.

FIG. 4. Photograph of a Type 1 splenic rupture. This spleen was removed from Case 9 of our series.

SYMPTOMATOLOGY

There is no one pathognomonic sign or symptom of traumatic rupture of the spleen. In almost every textbook of surgery Kehr's sign and Ballance's sign are prominently mentioned as diagnostic.[14] This has not been our experience, particularly with reference to Kehr's sign, which consists of referred pain in the left shoulder from irritation of the left diaphragm. A consideration of the literature

reveals that, while many writers mention it, few actually noted its occurrence in their own cases. Consideration of the pathologic findings in splenic rupture would indicate that it should be particularly found in instances of delayed hemorrhage. Ballance's sign is more frequently encountered. It consists of dullness to percussion in the left upper quadrant, left flank and even in the right flank. On the shifting of position, only the dullness in the right flank disappears.

TABLE IV

INCIDENCE OF COMMON SIGNS AND SYMPTOMS OF SPLENIC RUPTURE

In Order of Frequency

Generalized abdominal rigidity	13
Shock	12
Pain in left upper quadrant	12
Generalized abdominal pain	11
Rigidity in left upper quadrant	4
Initial unconsciousness	4
Pain on respiration	4
Dullness in left upper quadrant	4
Vomiting	3
Dullness in both flanks	2
Pain in left lumbar area	1

Table IV presents the incidence of the common signs and symptoms. The most frequently encountered findings are abdominal pain and rigidity, either generalized or localized to the left upper quadrant. Shock is another frequent finding, occurring in over half of our cases; however, it is not always present at the time the surgeon is called upon to give his judgment. We have noticed an initial shock which disappeared spontaneously. This has recurred in certain instances as the hemorrhage progressed. In other patients shock develops only as the effects of blood loss become otherwise evident. Initial loss of consciousness and vomiting were two of the less frequently encountered findings.

No discussion of traumatic rupture of the spleen would be complete without consideration of the syndrome of *delayed hemorrhage*. This was first described by A. H. McIndoe.[34] In 1932, McIndoe reported forty-five cases. Since then we have found thirteen more cases reported in the literature.[6,7,8,15,17,20,25,38,40,45,46,48] To this list we are able to add two more of our own.

This definite clinical picture merits more complete understanding, otherwise many a case will be overlooked. It is, in all probability, more common than the literature indicates. The syndrome may be described in the following manner: After an injury to the back, lumbar area, or abdomen, which frequently is not of great severity, the

patient suffers more or less pain, some nausea and perhaps even mild shock. After several hours these symptoms disappear completely, or at most, leave only a vague upper-abdominal discomfort. The patient then enters the so-called latent period and is able to be up and about.

TABLE V*

TIME ELAPSED BETWEEN INJURY AND ONSET OF SYMPTOMS

Time Interval	No. of Cases
Immediate	15
1–10 hours	3
11–48 hours	2
Delayed hemorrhage	2
Total	22

* This table shows the time interval elapsing between injury and the onset of symptoms leading to a diagnosis of ruptured spleen. The last two instances of delayed hemorrhage occurred eight days and five weeks, respectively, after injury.

The minimum length of the latent period should be forty-eight hours, as defined by McIndoe,[34] to differentiate it clearly from simple, slowly progressive hemorrhage which may take more than twenty-four hours to produce signs and symptoms. (Table v.) Also during the latent period the patient should be practically symptom-free. Latent periods as long as six months are reported in the literature. The latent period, after forty-eight hours or more, is suddenly terminated by the return of severe symptoms of intense abdominal pain and shock produced by the sudden release of a large amount of blood into the peritoneal cavity. This may occur so long after the unappreciated initial injury that the accident is even forgotten and the case considered a spontaneous rupture. During the latent period warning signals do exist. There will usually be dull pain on pressure over the left upper quadrant, a rapid pulse and a leukocytosis.

The entire syndrome is better understood when the underlying pathologic physiology is made clear. Types IV and V of splenic injury particularly predispose to its development. (Figs. 2 and 3.) Thus, a relatively moderate blow may be followed by a small subcapsular hematoma with slight maceration of the adjacent splenic pulp. The spleen appears unable to heal this type of injury. Following this, constant oozing into the subcapsular hematoma gradually causes it to expand, stripping the capsule from the spleen and further macerating the pulp. The hematoma swells to considerable size. Finally, a slight movement or strain is sufficient to cause it to rupture, releasing blood into the peritoneal cavity. A similar picture results when a small tear permits slow hemorrhage to occur. Sufficient time is thus allowed for the omentum, colon and stomach to wall off an area

about the spleen. A perisplenic hematoma is thus formed which may reach great size before the latent period is terminated by some slight strain or sudden movement sufficient to break down the barrier of adhesions.

Our two cases of delayed hemorrhage follow:

CASE 5. The patient, V. La F., a student at The Ohio State University, was admitted to the University Hospital shortly after an automobile accident, complaining of intense abdominal pain. Immediately after the accident he lost consciousness, but did not appear to be in shock at the time of admission.

Examination at the time of admission revealed: Temperature 98°F.; pulse 80; respirations 22; blood pressure 120/80. Respiratory movement of the left chest was decreased and left lower chest pain was experienced on inspiration. The abdomen showed marked generalized rigidity with marked tenderness in both upper quadrants. No masses were felt. The admission erythrocyte count was 5.06; the leucocyte count was 17,280.

The patient was admitted and placed under observation. By the next morning he felt well except for some residual soreness in the left upper quadrant. He was, however, kept in the hospital and in bed. Five days after admission his erythrocyte count was 3.42 and his leucocyte count was 21,900.

On the eighth hospital day the patient felt well and was allowed to sit in a chair. That evening almost immediately after returning to bed, he experienced severe abdominal pain and went into shock. His erythrocyte count at this time was 2.59 and his leucocyte count was 35,200. He was given a blood transfusion and taken to the operating room. At operation a ruptured, subcapsular splenic hematoma was found. This was treated by tamponade and suture. The patient made a complete recovery.

CASE 15. J. B., an eighteen-year old white youth, was admitted to the University Hospital complaining of severe abdominal pain, nausea and vomiting. Five weeks prior to admission he was in an explosion in which he received a blow to his left upper quadrant. He was observed in another hospital for two days, but upon revealing no signs of severe injury was discharged. He thereupon resumed his normal activity. Twelve hours prior to his second admission he developed severe generalized abdominal pain of a cramp-like nature. He became very weak, was nauseated and vomited.

Examination on admission revealed: Temperature 100°F.; pulse 110; respirations 20; blood pressure 90/50. The patient lay quietly in bed with both thighs flexed, and in great pain. The lungs were clear; the heart was normal. The abdomen revealed generalized rigidity and tenderness. The patient's erythrocyte count was 3.90 and the leucocyte count was 22,400.

Twelve hours after admission the patient was operated upon. A perisplenic hematoma was found. The spleen had a small peripheral laceration.

This was sutured and the splenic fossa packed. The patient made an uneventful recovery.

Before considering the diagnosis of splenic rupture, the subject of associated lesions should be considered. Wright and Prigot[48] found severe associated injuries in 50 per cent of their cases. In their series they encountered the following lesions, in descending order of frequency: Fractured ribs, fractures of the extremities, lacerated kidney, hemathorax with lacerated lung, cerebral complications, ruptured liver, ruptured urinary bladder and ruptured diaphragm.

TABLE VI*
ASSOCIATED INJURIES AND FATALITY

Type of Injury	Incidence	Fatality
Fractured ribs	5	None
Hemothorax	3	1
Fracture of extremities	3	None
Contusion of pancreas	1	None
Ruptured kidney	2	2
Retroperitoneal hemorrhage	1	1
Ruptured liver	2	1

* The associated pathologic lesions listed in order of frequency found in our group of 22 cases of splenic rupture. The effect of each complicating lesion on the outcome is indicated in the column at the right.

In Table VI the type and frequency of the associated lesions in our own series is recorded. In ten patients, or 45 per cent of the group, other injuries were present. The table also shows the effect of the associated damage on the outcome of the case. Undoubtedly, the presence of other injuries portends a poorer prognosis. However, the authors wish to make this point: Too often the associated condition, which may be more obvious but less serious, distracts the surgeon's attention. Only belatedly does he realize that the hemorrhage from a ruptured spleen is killing his patient. By the time this realization comes it may be too late.

An illustrative example is our Case 16, admitted in shock, with a fracture of the neck of the right femur and a fracture of the shaft of the left femur. The fractures were treated correctly, yet the patient died of hemorrhage from a ruptured spleen.

One further point should be made in connection with severe associated injuries. In reviewing our series we believe that too often when confronted with multiple severe lesions, the surgeon hesitates,

or is overwhelmed by the magnitude of the task before him. He temporizes, delays while treating the shock, and with the delay follows loss of the patient. Cases with trauma to other organs call for the most energetic intervention of all and even delay in treating the shock may result fatally.

Two cases in our series are again illustrative. Both had ruptured spleens and ruptured livers. (Table VI.) In one, operation was delayed thirty-six hours. The patient died. In the other operation was performed immediately after admission, while the patient was still in shock, with a blood pressure of 70/40. A splenectomy was accomplished and the liver sutured. The patient made an uneventful convalescence and was discharged on the twentieth postoperative day.

DIAGNOSIS

The diagnosis of traumatic rupture of the normal spleen may be very simple in the typical uncomplicated case, or extremely difficult when associated with additional injuries. The associated injuries may be so severe as to mask or even overshadow the symptoms of ruptured spleen. One of the cardinal symptoms, found constantly in reviewed cases, is that of abdominal pain either generalized or confined to the left side of the abdomen. This pain is usually very severe and may be out of all proportion to the suspected severity of the injury. History elicits some type of external violence. The patient presents severe progressive shock and may give a story of periods of unconsciousness. Breathing is usually thoracic in character, with respirations rapid and shallow. Ballance's sign may or may not be positive. On examination the surgeon is impressed by the rigidity of the abdomen which increases with the shock and the seriousness of the injury. The temperature early is subnormal, the pulse rapid and of poor quality, depending upon the degree of hemorrhage and shock.

In addition to the above clinical findings certain laboratory procedures may be done quickly and are of definite help. The leucocyte count in 80 per cent of the cases will be elevated above 10,000 and on repeat counts will be found to be rising. The erythrocyte count and hemoglobin may show a slight increase above normal at first (Table VII) but on repeated counts will be found to be falling rapidly. The falling drop method of Scudder for determining early hemoconcentration in incipient shock can be run quickly and will prove most helpful in making an early diagnosis.

Webb[45] has stressed the importance of a roentgenogram of the abdomen. This will frequently show elevation of the left diaphragm. The authors agree with this view completely. The procedure takes but little time and can be most helpful, particularly in cases of delayed hemorrhage.

TABLE VII

BLOOD AND URINARY FINDINGS ON ADMISSION IN OUR TWENTY-TWO CASES OF SPLENIC RUPTURE

	No. of Cases
Red Blood Cells	
Above 4.00	7
Above 3.00	9
Less than 3.00	3
White Blood Cells	
Above 20,000	7
Above 10,000	9
Less than 10,000	2
Red Blood Cells in Urine	5

Wright and Prigot[48] stress the use of paracentesis to determine abdominal hemorrhage. This is undoubtedly of value in certain instances, but we do not believe that its routine use, as suggested by them, is necessary or justified.

Burke and Madigan[10] suggest the intravenous injection of thoratrast. This is an ingenious method and in a limited number of obscure cases may be most helpful. The reticulo-endothelial elements of the spleen phagocytize particles of the contrast medium. But a delay of at least two hours is necessary to interpret the test.

The conditions from which ruptured spleen must be differentiated are mainly acute abdominal emergencies which require prompt surgical intervention. Perforated peptic ulcer, mesenteric thrombosis, ruptured kidney or liver, ruptured tubal pregnancy may all be mentioned. Of all these, rupture of the left kidney is the most likely to cause confusion. Whenever this enters as a possibility, analysis of the urine and an intravenous pyelogram can be done quickly and should serve to make the differential diagnosis.

We wish to stress the importance of early diagnosis. Fifty-one per cent of patients with ruptured spleens die within one hour following injury. We realize that but few of these cases may have the opportunity for adequate treatment, due to the severity of their injury and the rapidity of its course. The mortality rate in the remaining 49 per cent may be lowered by prompt diagnosis and adequate treatment. All patients with a history of abdominal injury should be carefully examined, re-examined and observed, bearing in mind the probabil-

ity of injury to the spleen, and the seriousness of the situation should this exist. The surgeon should realize that an error in diagnosis or a tendency to minimize the situation may be fatal to the patient.

TABLE VIII

ADMISSION AND PREOPERATIVE DIAGNOSES IN OUR SERIES

Ruptured spleen	6
Ruptured intra-abdominal viscus	4
Intra-abdominal hemorrhage	4
Possible ruptured spleen	2
No diagnosis	2
Fractured ribs—Intra-abdominal injury	1
Acute suppurative appendicitis	1
Laceration of lung	1
Coronary occlusion	1
Total	22

Finally, the importance of making the correct diagnosis preoperatively cannot be overemphasized. Table VIII lists the preoperative or admission diagnoses in our series. In only six cases, or 27 per cent of the series was the correct diagnosis made preoperatively. Of these only one died, giving a mortality of 16 per cent. Of the remaining sixteen cases incorrectly diagnosed, or undiagnosed, six died resulting in a mortality of over 37 per cent.

TREATMENT

A brief backward glance at the early attempts at surgery for traumatic rupture of the spleen is interesting. The first successful splenectomy for splenic rupture was performed in 1892.[5] By 1900, Bessel-Hagen[4] reported thirty-seven patients operated upon with twenty recoveries or a mortality of 46 per cent. Lotsch,[30] in 1908, reported 138 operative cases with a mortality of 37 per cent. With general improvement in surgical procedures and a more widespread awareness of the occurrence of ruptured spleen, this rate has gradually lowered. By 1922, Buxton,[12] reviewing the cases reported up to that time, found a mortality rate of 28.8 per cent of all patients operated upon.

The treatment of traumatic rupture of the spleen or of rupture with delayed hemorrhage is surgical. Conservatism has but small place in the treatment of ruptured spleens. Wright and Prigot[48] record the mortality in conservatively treated cases, in those refusing treatment, or in those undiagnosed, as from 90 to 100 per cent. They also state that 51 per cent of patients with a ruptured spleen die within

one hour following their accident. We may conclude from these statistics that *surgical treatment must be prompt and energetic.*

Patients presenting shock should be treated by shock combatting methods such as intravenous fluids, plasma, or by slow drip blood transfusion *during* surgery. It is impossible to expect to alleviate shock in a patient who is hemorrhaging from a large tear in the spleen. If operation is delayed until the patient recovers from shock, but few ruptured spleens will come to operation. We cannot stress too strongly that shock is not to be considered a contraindication to surgery in this condition.

Each patient should be treated individually. The choice of the anesthetic agent, depending upon facilities at hand, should be made with regard for the safety of and suitability for each particular case. Many patients are started under local and supplemented with inhalation anesthesia.

On opening the abdomen, in nearly all cases of ruptured spleen, a large amount of free blood is found. This blood should be aspirated into a sterile container, filtered and then reinjected into the patient's vein. Autotransfusion[26,28] originally advocated in the literature by Kraft, in 1931, is but rarely mentioned yet will be found to be life-saving in many cases. No ill effects occur since the blood is not contaminated. Post-transfusion reactions do not occur as they sometimes do in using donor blood, while the free blood in the abdominal cavity affords a means of prompt transfusion to each case. One of us (W. D. N.) has used this method most successfully.

The ruptured spleen should be quickly inspected and frequently may be grasped with the hand to control bleeding before clamping the splenic pedicle. From careful consideration of our own series and a review of many other cases, we have concluded that suture or tamponade has no place in the treatment of the ruptured spleen. *Any fractured spleen should be removed.* When the splenic vessels are suitably ligated any danger from hemorrhage has been obviated. Due to the consistency of the organ it is most difficult to suture the spleen satisfactorily. Tamponades may control hemorrhage while *in situ*, but their removal is frequently followed by recurrent or even by delayed hemorrhage. We have no assurance that a sutured spleen will heal satisfactorily or that delayed hemorrhage will not occur.

The few who advocate suture and packing are at variance with the majority opinion on the subject. Dretzka[18] writing of civilian practice and Gordon-Taylor[21] reporting on war injuries to the spleen, both hold that suture is satisfactory for gunshot and penetrating

wounds. However, Jolly[27] also writing of war surgery, is definitely against such a procedure.

The operation should be done as quickly as possible with adherence to the fundamental principles of surgery. Hemostasis must be

TABLE IX*

TREATMENT BY SPLENECTOMY IN RELATION TO MORBIDITY AND MORTALITY

Case No.	Associated Lesions	Mortality	Stay in Hospital—Days
1	Contusion of pancreas	Recovered	35
3	Pneumonia	Recovered	89
4	Hemothorax	Recovered	17
7	Fractured extremities	Recovered	12
8	None	Recovered	21
9	None	Recovered	30
11	None	Recovered	14
12	Fractured ribs	Recovered	22
13	None	Recovered	8
18	Ruptured liver	Recovered	24
22	Ruptured diaphragm and lung	Died	1
11	7	1	27

* This table lists the results in eleven cases treated by splenectomy. The associated lesions are included to indicate the severity of the injury. Morbidity is measured by the length of stay in the hospital.

secured; a rapid but careful search for additional injuries such as perforation of the stomach or bowel in penetrating wounds; and rupture of the diaphragm, kidney, or liver, in crushing injuries must be made. Wounds not contaminated are closed without drainage.

Postoperatively, these patients are treated as any other surgical patient in shock or after hemorrhage. Blood transfusions should be given. We have found repeated small transfusions given frequently of more benefit than the same amount of blood given as a larger transfusion. Fluids by vein and clysis, and blood plasma also have their place in postoperative care. Morphine should be used freely to produce rest and relaxation.

The data in Tables IX, X, XI, and XII summarize our findings in regard to treatment. Table IX reveals that eleven of our twenty-two patients were treated by splenectomy. In this group there was one death. This is a mortality of 9 per cent. The patient who died was long delayed in securing medical attention and was practically *in extremis* when operated upon. Table X shows that four patients were treated by suture or packing. Of these none died. The mortality

rate for the fifteen operated cases is thus 6.6 per cent. This is an exceptionally low figure. The average reported in the literature is about 25 per cent. Yet, when this is compared with the mortality

TABLE X*

TREATMENT BY SUTURE AND PACKING IN RELATION TO MORBIDITY AND MORTALITY

Case No.	Associated Lesions	Mortality	Stay in Hospital—Days
5	None	Recovered	33
14	None	Recovered	33
15	None	Recovered	22
19	None	Recovered	62
4	0	0	37

* This table presents the results of more conservative therapy in the management of four cases of splenic rupture. There were no severe associated lesions.

TABLE XI*

MORBIDITY AND MORTALITY WHEN NO TREATMENT IS ATTEMPTED

Case No.	Associated Lesions	Mortality	Stay in Hospital—Days
2	Ruptured kidney	Died	1
6	None	Died	2
10	None	Recovered	36
16	Ruptured liver	Died	2
17	Ruptured kidney	Died	1
20	Hemothorax	Died	2
21	Rheumatic heart	Died	11
7	5	6	8

* This table shows the results obtained in 7 cases of splenic rupture when no treatment other than observation or medical management was attempted.

rate of 85 per cent in the untreated group, it is convincing that conservative or nonoperative treatment has little if any place in the management of traumatic rupture of the spleen.

A comparison of Table IX and X as summarized in Table XII presents the difference in morbidity between those patients treated by splenectomy and those treated by suture or packing. As measured by length of hospital stay, the patients treated by splenectomy showed a lower morbidity than those treated by other operative measures—twenty-seven days as compared to thirty-seven days.

The question of spontaneous cure usually arises in any discussion of treatment. Some surgeon can usually be found who will recite his

case of ruptured spleen in which the patient made a spontaneous recovery. To deny categorically that such is possible would be imprudent. We even include one such case in our group.

TABLE XII
SUMMARY OF RESULTS OBTAINED BY THE VARIOUS TYPES OF TREATMENT IN RELATION TO MORBIDITY AND MORTALITY

Treatment	No. of Cases	Mortality	Stay in Hospital—Days
Splenectomy	11	1	27
Suture and packing	4	0	37
No operation	7	6	8

CASE 10. H. S., a twenty-eight-year old white male, was admitted to the hospital complaining of pain in the left upper quadrant after being struck by an automobile. He did not lose consciousness but immediately after injury noted pain in the left lower chest and left upper abdomen.

Examination revealed: Temperature 101°F.; pulse 90; respirations 48; blood pressure 106/54. There was marked tenderness in the left upper quadrant and flank and dullness to percussion in the left flank. Admission erythrocyte count was 4.69; the leucocyte count was 32,100.

Immediately after admission the patient went into shock. His blood pressure dropped to 80/50. He was treated conservatively and made rapid improvement. On his eleventh hospital day he was discharged. On the way home, while still in a taxi, he again suffered severe abdominal pain and collapsed. He was immediately returned to the hospital, arriving in shock with a blood pressure of 84/60. The signs and symptoms on examination were identical with those of his previous admission. Again he was treated conservatively and gradually improved. He was finally discharged on his twenty-fifth hospital day.

This case illustrates well the matter under discussion. In the first place, if operation is not performed, the surgeon cannot be sure that the patient had a ruptured spleen. Second, having once grasped the full implication of delayed hemorrhage, the surgeon cannot quite be sure but that the patient will have a recurrence of the bleeding at a later date. If, in the case presented above, the patient had gone to another surgeon at a different hospital after his first discharge, the original surgeon would still be talking about his case of spontaneous recovery. Even at present there is no certainty but that two days or two months after his second discharge, the patient will not again have a delayed hemorrhage.

To summarize then, we conclude that the treatment of traumatic rupture of the spleen is always surgical and that the operation of choice is splenectomy.

COMPLICATIONS

The most commonly mentioned postoperative complication is intestinal obstruction, due either to paralytic ileus or adhesions from trauma to the intestines and mesentery. Three such instances occurred in our group. Disruption and infection of the wound may occur as a result of injury to the tail of the pancreas, or to undue haste in wound closure. One of our patients suffered from this complication. Postoperative lung complications are frequent because of associated injury to the chest. In our series there were three postoperative pneumonias in addition to the hemothoraces already listed. These and other complications must be recognized early and suitably treated.

POSTOPERATIVE RESULTS OF SPLENECTOMY FOR TRAUMATIC RUPTURE OF THE SPLEEN

It is significant to note that but few cases have been carefully studied postoperatively after having had a splenectomy for splenic rupture. Yet here is a most fertile field in which to add to our all too hazy knowledge of the normal function of the spleen. Many reports have been written on the postoperative results of splenectomy for pathologic conditions of the spleen,[31,45,47] but here in a group of vigorous, healthy individuals suddenly deprived of a normal spleen, but little investigation of real value has been accomplished.

The material embodied in this section may be considered as a preliminary report of work in progress by one of us (L. C. R.).

In 1924, Pfeiffer and Smyth[37] made one of the few reports specifically dealing with the late results of splenectomy for traumatic rupture of the spleen. Before that Hitzrob,[23] in 1918, published a paper on "The Effect of Splenectomy on Normal Individuals and in Certain Pathological Conditions."

The resultant effects which these authors and others generally attribute to such splenectomies may be listed as follows: (1) An anemia which gradually returns to normal in from three to four months; (2) an increased resistance to destruction of the red cells; (3) an increased output of iron in the stools; (4) an increased total blood cholesterin; (5) no change in opsonins and agglutinins; (6) pronounced enlargement of the superficial lymph glands; (7) ease of

fatigability, and (8) lowered number of blood platelets, considering 600,000 as normal.

These results are based on four cases investigated at periods

TABLE XIII

LATE POSTOPERATIVE FOLLOW-UP OF FOUR CASES OF TRAUMATIC RUPTURE OF THE SPLEEN TREATED BY SPLENECTOMY

Case No.	Age at Splenectomy Yrs.	Interval Since Splenectomy Yrs.	Hematology					Growth	Increased Incidence of Infection
			Hemoglobin	Red Blood Cells	White Blood Cells	Platelets	Differential		
1	14	2	14.5	4.90	7,200	800,000	Normal	Adequate	None
12	8	7	14.	4.00	7,500	848,000	Normal	Adequate	None
9	20	3	14.5	4.90	6,250	690,000	Normal	Adequate	None
11	12	3	13.5	4.25	6,500	700,000	Normal	Adequate	None

ranging from one year and nine months to four years postoperatively. Other observers have attributed a failure of proper growth and a decreased resistance to infection to the removal of a normal spleen.

Our studies on this subject are based on six patients who received splenectomies for splenic rupture. The period postoperatively ranges from two to seven years. Four have been partially investigated, while two have only had a preliminary interview. We are most fortunate in having the assistance of the hematologic laboratory of Drs. C. A. Doan and B. K. Wiseman in carrying out this work.

Table XIII gives certain of our findings in our first four cases. The columns headed "Growth" and "Infection" indicate that according to clinical tests there was no deficiency of the one or increased evidence of the other. Another fortunate circumstance, as shown in the table, is that all these cases were adolescent at the time of splenectomy.

Specifically our investigation thus far shows that all four patients have a normal hematologic picture. None showed either anemia or thrombopenia. All four showed normal growth. None showed any increased incidence of infection. None of the four had any appreciable enlargement of the superficial lymph glands. All four had normal basal metabolic rates ranging from minus 2 to plus 8. In all the plasma iron was within normal limits. None admitted any decrease in energy or ease of fatigability. All were actively engaged in sports and extracurricular scholastic activities.

Three, however, have shown one significant finding. When their blood volumes were investigated they were found to be normal. How-

ever, after the injection of adrenalin their blood volume did not increase as does that of the normal individual. It remained remarkably constant. Furthermore their blood pressures showed only a 6 to 8 millimeter of mercury increase. They did, however, show an increase in pulse rate. The fourth case has not yet been thus investigated. Therefore, to the question, "What is the effect of splenectomy on a normal individual?", we must perforce answer, "We have found none." So far as health and growth are concerned, the spleen appears to be an unnecessary organ.

One other late result of traumatic rupture of the spleen should be mentioned. In 1892, Albrecht[1] reported a case which at autopsy was found to have no primary spleen but numerous small growths of splenic tissue on the greater omentum, the diaphragm and in the pouch of Douglas. There was no history of trauma or of splenectomy. These growths may have been multiple accessory or ectopic spleens which have been found in a high percentage of necropsies. Albrecht, however, reported them as splenic autotransplants.

In 1910, von Kuttner reported a case, necropsied four years after splenectomy, in which from eighty to one hundred splenic growths were found scattered throughout the abdomen. These could hardly have been accessory spleens. Since then Foltin,[19] von Stubenrauch,[44] Oltman,[44] Lee,[29] Kupperman,[42] Shaw and Shaft[39] in Europe; and Buckbinder and Lipkoff[9] in this country, have each reported one case of splenic autotransplants. Buchbinder and Lipkoff have suggested the name *splenosis* for this condition.

In 1920, Marine and Manley[33] successfully made autotransplants of splenic tissue to the abdominal muscles of rabbits. In 1936, Perla[36] produced peritoneal splenic autotransplants in rats.

In our own experimental work we have produced peritoneal splenic autotransplants in three out of six rabbits. These were not recognizable earlier than six months after implantation. Figures 5, 6 and 7 present the gross appearance and microphotographs of the transplants in one such experimental splenosis. Our technic has been to splenectomize the rabbit under conditions of strict asepsis. The spleen is then aseptically macerated to the consistency of a thick paste. Small portions of this, no larger than a pinhead, are then seeded throughout the abdomen.

From this experimental study we conclude that splenosis is a rare but definite and serious late complication of splenic rupture. The seriousness lies in the fact that the splenic tissue may be simultane-

ously implanted on two adjoining loops of bowel. This may, in time, provide the basis for an intestinal obstruction. One such case has been reported. Another untoward result of splenosis could conceiv-

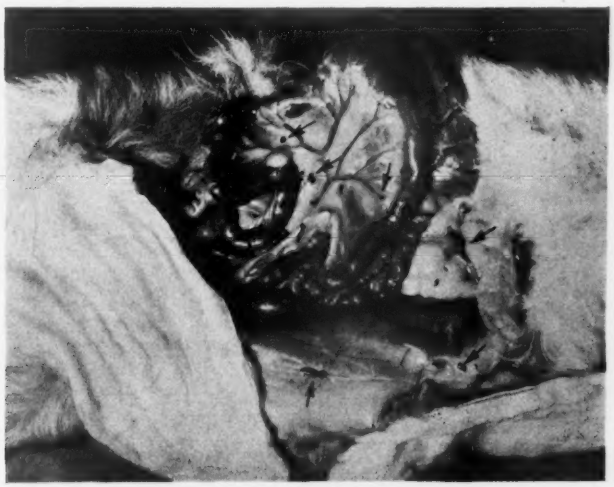

FIG. 5. A photograph of the intestine, mesentery, and parietal peritoneum of a rabbit showing numerous areas of autoplastic splenic transplants.

FIG. 6. Microphotograph, low magnification, showing a portion of a splenic autotransplant attached to the serosa of the intestine of a rabbit.

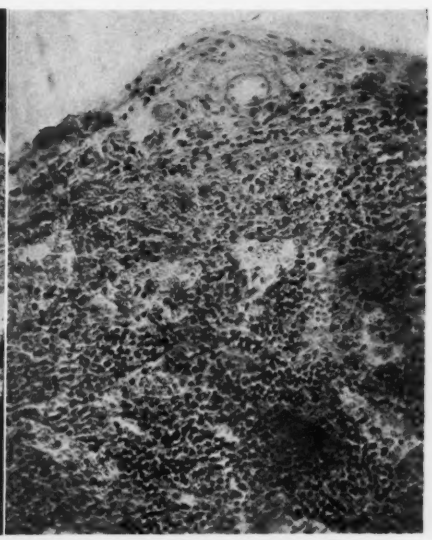

FIG. 7. Microphotograph, high magnification, of the same specimen as Figure 6, showing cellular detail of splenic autotransplant.

ably be the development of an abnormal hemoclastic activity by the transplanted splenic tissue.

CONCLUSIONS

Traumatic rupture of the normal human spleen is not as rare as is commonly thought. Its incidence, both real and apparent, is increasing in civilian life and is high in war injuries. The spleen is the most frequently injured of all the abdominal viscera. The types of injury most frequently causing this lesion, aside from war injuries, are automobile accidents and falls. Five types of pathologic rupture are recognized, each leading to a definite clinical picture. Of these the syndrome of delayed hemorrhage is the most frequently overlooked.

Associated severe lesions occur in about 50 per cent of the cases. These materially increase the likelihood of a fatal outcome and may call for rapid surgical intervention.

There are no pathognomonic signs or symptoms of splenic rupture. Generalized abdominal rigidity and tenderness, or the same more localized to the left upper quadrant are encountered in the majority of cases. Shock is also most frequently noted. These clinical findings together with certain simple laboratory tests enable the surgeon to make the diagnosis with certainty. The laboratory tests of greatest value are: repeated determinations of the hemoglobin and erythrocyte count, the Scudder falling drop test for hemoconcentration and a flat plate of the abdomen showing an elevated left diaphragm.

The treatment of traumatic rupture of the spleen is always surgical. Splenectomy is the operation of choice. Operation should not be delayed in an effort to combat shock. This should be treated while the operation is in progress. Autotransfusions are recommended.

Ultimate ill effects subsequent to absence of the spleen after splenectomy for splenic rupture are negligible. We have been able thus far to determine no ill effects. Splenosis or the occurrence of splenic autotransplants within the peritoneal cavity is a real but rare late complication.

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DISCUSSION

W. L. ESTES, JR. (Bethlehem, Pa.): I believe the Association is to be congratulated on the delightful paper that Dr. Curtis and Dr. Roettig have prepared, and its presentation by Dr. Roettig.

We have been interested in various phases of this particular problem. Our experiences differ somewhat in the question of incidence of abdominal trauma, and also of lacerations and rupture of the spleen. We find that nonpenetrating trauma of the abdomen is encountered rather infrequently, and to many the diagnosis and treatment are not always familiar. For that reason, therefore, we believe that this is a very valuable paper, having brought to our attention various pitfalls in the possibilities of incomplete and uncertain diagnosis, and having emphasized the difference in accuracy of proper treatment and the end results. This very extensive report of Dr. Curtis is most timely on the rupture of the spleen representing a lesion that requires prompt recognition and prompt surgical intervention for a successful outcome.

Our experience has been, statistically, that of about 415 consecutive injuries admitted to our clinic. There were thirty-four of abdominal trauma, and of another group of ninety-four cases of abdominal trauma, there were seven instances of rupture of the spleen.

These cases of rupture of the spleen we have found more or less helpful to divide into three groups:

1. *Cases of severe multiple trauma,* which include rupture not only of the spleen but also of the diaphragm, liver, intestine or kidney, fracture of ribs and extremities, or lacerations of lung. These terminate fatally so quickly that very seldom is there an opportunity to apply treatment that might be effective. The trauma is so great and extensive that death is inevitable.

2. *Simple rupture of the spleen with a recognizable symptom complex.* That, again, I think Dr. Roettig dwelt upon very nicely and satisfactorily. Splenectomy is the treatment of choice, and as Dr. Roettig has shown, can be performed with a surprisingly low mortality with adequate preoperative and postoperative care.

3. *Cases with delayed symptoms or delayed rupture, with obscure or atypical manifestations,* also referred to by Dr. Roettig. These, unfortunately, are not uncommon and present a problem in diagnosis that may be very difficult to solve in time to save the patient.

Our experience with traumatic rupture of the spleen has been extremely limited. Five of these were the result of motor accidents and a fall, and one a coasting accident, and I am wondering whether that may not be a factor in the experience and incidence as between Drs. Curtis and Roettig and ourselves. I believe that in the congested, heavily populated areas, where they are many motor accidents, this injury is probably going to be found to be much more common than perhaps in the less heavily populated districts because of the high incidence of severe motor accidents.

Of these seven, three had severe multiple injuries and died within sixteen hours of the accident with no surgical treatment. Four were splenectomized. Two had uncomplicated ruptures of the spleen and survived. Two died, one as a result of concomitant contusions of the liver and intestine with terminal mesenteric thrombosis; the second had fracture of the ribs with laceration of the lung.

This in general, if any conclusion can be drawn from so few cases, corroborates Connor's dictum that mortality in rupture of the spleen depends upon the severity of the associated lesions, and that the only effective treatment is prompt splenectomy in the properly prepared patient, by transfusions, plasma, etc. It seems likely, however, with the various blood substitutes now on hand for immediate use in emergencies, that the severe complications of splenic rupture may be more successfully handled, particularly when hemorrhage is the major lethal factor.

We have also been interested in this group of cases of delayed diagnosis. Of definite interest have been various measures proposed from time to time

to aid in the diagnosis of delayed rupture. Dr. Roettig has very well stressed the point brought out by McIndoe that the *x*-ray has too seldom been used. With splenic rupture, in a simple flat plate of the abdomen McIndoe observed increased density in the left upper quadrant with elevation of the left diaphragm and displacement of the stomach to the right. Webb has reported similar findings.

Pendergrass has suggested that a radiograph taken after administering a Seidlitz powder will better outline the stomach and reveal the pressure deformity in the fundus that occurs with a large perisplenic clot. And finally, Burke and Madigan have likewise reported the intravenous use of thorotrast to diagnose rupture of the spleen. An abdominal film taken four hours after injection will demonstrate perisplenic extravasation of dye when splenic rupture has occurred.

We believe that the successful treatment of a traumatic rupture of the spleen would seem to depend upon the severity of concomitant lesions, the prompt combating of blood loss with equally prompt splenectomy, and early diagnosis of delayed rupture or rupture with delayed or slow hemorrhage, so that the indications for splenectomy may be recognized before it is too late.

G. Gavin Miller (Montreal, Canada): I am afraid, that I have not very much to add. There is one sign we see at times which is that of pain in the shoulder, and I think sometimes the gas bubble in the stomach with a flat plate will be shown on a straight *x*-ray examination. I certainly have never believed that one should do elaborate *x*-ray tests.

We have eight patients in our series and none of them died. They were all treated differently and they were all treated at different times. These are all cases with no multiple injuries. The patients with multiple injuries are apt to die rather promptly, and when the autopsy is done by the coroner we have no information on that series. In the cases in which we have operated upon the patients we find we have had no mortality.

I have been impressed much more by the danger of missing the diagnosis which may be a ruptured bowel. I think that early operation, splenectomy, with suitable transfusions, is a comparatively safe procedure, but I think delay in which there might be a ruptured bowel is fraught with great danger. I think that a ruptured bowel is an infinitely more dangerous condition, especially if missed, than splenectomy; so it has been our rule, where there is any doubt about the diagnosis, to explore; and I think with a suitable local anesthesia, one need not be particularly afraid of opening the abdomen in the presence of a certain amount of shock and a certain amount of hemorrhage.

Frederic W. Bancroft (New York, N. Y.): May I mention one other diagnostic point that I think is sometimes of value? We had a child brought into the hospital this year with rather an indefinite history. By flat *x*-ray plate, we discovered a space between the cardia and the diaphragm. We

utilized that. We gave her a little barium by mouth, put her in Trendelenburg position, and found a space of an inch between the cardia and the diaphragm. I think that is a method of determining in the slow type the presence of blood.

Byrd C. Willis (Rocky Mount, N. C.): I want to say a few words about an instrument I use for collection of blood.

This is an apparatus we found very useful, and a very simple one to arrange. In the question of collecting the blood, we found this problem: If you use ordinary suction, you soon clot the blood and block the holes. You have to have a special tip. We have a glass tip and a wire to protect it, and we cover it with gauze. We have a section with the suction union at the top. You have your citrate container on top of that, and you have your air vent at the top.

You have at the bottom of the bottle your transfusion needle that you run right into your patient. You suck the blood in and when you have a sufficient quantity in, you open it up and let the blood run into the patient's vein. We found it of very great assistance in appendix ruptures and in tears in the mesentery, and I have recently used it (I will probably be severely criticized) in a case of evisceration in a Negro, who had six perforations of the bowel. I am convinced there was no leakage in the cavity because the eviscerated bowel had the perforation, but still I used the blood and this Negro survived.

I believe that the time is probably coming when we are going to stop throwing away the most valuable thing the patient has, and that is his own blood.

We have had eleven cases of ruptured spleen, and I should like to call attention to a type of injury that I have not seen quoted before. A man was grabbed around the abdomen by his friend, lifted up, and he did not pay any attention to it. This was the afternoon before his admission. He was admitted into the hospital and the internist made a diagnosis of acute appendicitis because of pain in the lower left quadrant. But he did give one significant symptom, that he had a pain in the left shoulder.

I operated upon the man for acute appendicitis, making a McBurney incision. When I opened the peritoneum there was a flood of blood. If I had not obtained the history of pain in his left shoulder, I would have been at a loss to know where this hemorrhage was coming from. I immediately closed the McBurney incision, made a left rectus incision, and found a transverse rupture of the spleen.

SPRAINS AND SEPARATIONS OF THE INFERIOR TIBIOFIBULAR JOINT WITHOUT IMPORTANT FRACTURE

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WHEN the term, "sprained ankle," is used, reference is usually being made to stretching or tearing of the external lateral ligament of the ankle. In view of the rather complex ligamentous structure of the ankle and tarsus, it would be more than a little strange if all sprains in this locality were confined to the external lateral ligament. As a matter of fact, several clinical entities may be recognized by careful examination. By far the most important and most serious ligamentous injury about the ankle is sprain of the anterior inferior tibiofibular ligament, with varying degrees of lateral luxation of the ankle, but unaccompanied by an important bone injury. In the severer grades of this lesion, failure to recognize its true nature and institute proper treatment results in disability all out of proportion to the apparent seriousness of the injury. Injury to the inferior tibiofibular ligament may occur alone, but it is usually associated with sprains or tears of the internal or external lateral ligaments.

INCIDENCE

Sprain or rupture of this ligament, with consequent spreading of the mortise, is universally recognized as a fairly common complication of ankle fractures, but its occurrence without an important fracture is overlooked by most authors. Campbell[1] refers to this injury but states that it rarely occurs without accompanying fracture. Keys and Conwell[2] mention it briefly, but regard it as rare. Only two references[3,4] to this condition could be found in the current periodical literature. Older textbooks disregard it entirely.

However, Böhler[5] considers it a relatively common lesion and discusses the mechanism and treatment at considerable length. Watson-Jones states that sprains of the internal lateral ligament may be accompanied by tearing of the tibiofibular ligament. Thorndike,[6] in his monograph on athletic injuries, says that damage to the

tibiofibular ligament is sustained more frequently than external lateral ligament injuries. In our experience, minor degrees of this lesion have occurred about as frequently as the usual "sprained

A B

FIG. 1. A, separation of tibiofibular joint by external rotation. B, roentgenogram showing displacement possible after novocainization of injured ligament.

ankle" and severe tears with lateral subluxation of the ankle are by no means infrequent. We have seen it most frequently as a result of athletic injuries—football, basketball, skiing, and the like—all having been responsible. As might be expected, it has generally affected patients in the second decade, although no age is exempt.

ANATOMY

The anatomy of the distal tibiofibular joint[7] is comparatively simple. The articulation belongs to the syndesmosis group which includes those joints in which contiguous bony surfaces are united by an interosseous membrane allowing a slightly movable articulation. This syndesmosis is formed by the rough convex surface on the medial side of the fibula and a rough concave surface on the lateral side of the tibia.

There are four ligaments in relation to this joint. The anterior ligament is a flat, triangular band of fibers which extends obliquely downward and lateralward between the adjacent margins of the

tibia and the fibula on the front aspect of the syndesmosis. The posterior ligament, smaller than the preceding, is disposed in a similar manner on the posterior surface of the joint. The inferior

A B C

FIG. 2. A, separation of tibiofibular joint by internal rotation. B, roentgenogram after novocainization of ligament. Note separation possible. Arrow indicates small avulsion fracture. C, lateral roentgenogram showing slightly displaced fracture of posterior lip.

transverse ligament lies in front of the posterior ligament, passing transversely across the back of the joint. The fourth ligament, associated with the joint, is the interosseous ligament which is continuous with the interosseous membrane and which constitutes the chief bond of union between the bones. A small amount of motion normally occurs in the tibiofibular joint as the astragalus moves in the mortise joint during plantar and dorsal flexion. This is due to an anatomical peculiarity. The body of the astragalus is much wider anteriorly than it is posteriorly. Hence as the ankle is dorsiflexed the wider portion of the astragalus enters the mortise and a slight normal separation occurs between the tibia and fibula. Conversely, the joint narrows as plantar flexion occurs. The nature of this motion is not clearly established, but it probably consists of slight external rotation of the fibula on its long axis, and possibly a gliding motion of the fibula on the tibia. There is a normal wide variation both in the

FIG. 3. A, chronic sprain of inferior tibiofibular joint. (Duration of symptoms two years.) B, roentgenogram of unaffected side. C, marked displacement possible after novocainization of ligament. D, injured ankle after autogenous bone graft, with complete relief of symptoms.

width of the joint fissure between the tibia and fibula, and also in the range of motion. It is, therefore, not possible to define the normal width of the joint or the normal amount of separation occurring during dorsiflexion, except by comparing the two sides. Neither age, sex, nor habitus seems to determine the type of joint a given individual has.

MECHANISM

Campbell[2] states that injury to the tibiofibular ligament may be the result of either direct or indirect violence. In our experience, the lesion has always occurred as a result of rotation of the leg on the fixed foot. The rotation may be either internal or external. Böhler[5] explains the occurrence of this lesion as follows: "If external rotation occurs on the fixed foot, the astragalus acts as a lever which is rotated inward in the ankle mortise stretching or tearing the tibiofibular ligament; or the fibula may be held in place by the external lateral ligament and only the tibia rotates. In this instance, the anterior outer angle of the tibia, to which the ligament attaches, is usually avulsed. If the leg is internally rotated on the fixed foot, the ligament between the medial malleolus and the astragalus is ruptured and then the tibiofibular ligament. Often a small fragment is torn off the posterior tibial malleolus, and if the force is continued, a torsion fracture of the upper end of the fibula occurs."

SYMPTOMATOLOGY

The symptoms naturally vary somewhat with the severity of the injury. In the less severe cases, pain in the ankle, swelling, and exquisite tenderness sharply localized over the tibiofibular joint, are found. In the more severe cases, with lateral subluxation, there is practically always inability to bear weight. While the findings on inspection are by no means as marked as in external lateral injury, the disability is much greater.

DIAGNOSIS

In a patient with the above symptoms, whose roentgenogram is reported negative, tibiofibular ligament injury should be strongly suspected. Localized swelling and tenderness over the anatomical location of the tibiofibular joint and absence of swelling below the malleoli make the diagnosis quite certain. If the ligament is ruptured or its bony attachment avulsed, the ankle will be loose. However, due

to reflex spasm of the muscles about the ankle, it may not be possible to demonstrate laxity in the mortise joint until the pain and spasm have been overcome. This may be accomplished by the injection of 2 per cent novocain directly into the injured ligament. After a few cc. of the novocain solution have been injected, it may be possible to withdraw old blood into the syringe, showing the presence of a hematoma. As soon as pain and spasm have been overcome, abnormal lateral mobility in the ankle can be demonstrated. This is accomplished by grasping the lower leg in one hand and the heel in the other. The astragalus can now be moved laterally and medially, and felt to strike against the internal malleolus.

As before mentioned, it is wise to test the uninjured side for comparison, since the normal range of motion varies in different individuals, much as it does in the inferior radio-ulnar joint.

Radiographic Appearance. Since the displacement is a matter of millimeters, routine radiographic examinations are usually negative, although comparison with the sound side may disclose a difference in the width of the joint fissure. After anesthesia, however, the astragalus may be pushed laterally against the external malleolus, in which event not only will the joint space appear widened, also the space between the astragalus and internal malleolus may be increased.

A widened space between the astragalus and the internal malleolus is evidence of a tear of the internal lateral ligament, which is produced by abduction. If the injury to the tibiofibular ligament is by external rotation, the internal lateral ligament need not be torn; if the injury is by internal rotation, only the anterior portion (the tibionavicular ligament) of the deltoid ligament need be affected. In either event, wide separation between the internal malleolus and the astragalus will not occur, and the latter finding is, therefore, not necessary to make a diagnosis of tibiofibular diastasis. It goes without saying that in attempting to show separation, the *x*-ray tube must be properly centered, otherwise the oblique rays produce a misleading distortion of the joint.

In the chronic case, the findings are generally less marked, but there is a history of one severe episode with slow recovery and followed later by milder acute exacerbation due to trivial traumas. A painful snapping in the joint is often noted by the patient and may be elicited by the examiner. It is our impression that many and perhaps most of the cases of so-called "chronic sprain of the ankle" really represent unrecognized injuries to the inferior tibiofibular ligament.

TREATMENT

In those instances in which no displacement can be produced under local anesthesia and where the patient is able to walk, a zinc-gelatin boot provides sufficient compression and support. It is repeated at two-week intervals until the pain, swelling and tenderness have abated.

In cases in which subluxation can be detected, reduction and fixation in plaster of paris must be carried out to prevent the ligament healing in a relaxed condition. Campbell[2] suggests the use of a padded cast, windows being cut over the malleoli and felt pads inserted to secure a compressing effect.

In our hands, the use of a nonpadded walking cast, a la Böhler, has proved satisfactory. Under local anesthesia, as much of the swelling as possible is removed by massage and kneading. A U-splint is then smoothly applied to the lateral surface of the leg and ankle and bandaged with gauze. As setting occurs, marked pressure is exerted on the malleoli, the thenar eminences of the hands being used to distribute the pressure over a considerable area. A posterior plaster mold is then bandaged in place and the cast completed by a circular plaster bandage. Next a walking iron is incorporated in the cast and weight bearing permitted. The cast is allowed to remain from four to six weeks when it is removed and a zinc-gelatin boot applied until all tenderness and swelling have subsided.

In chronic cases, radical treatment may be necessary in order to effect a cure. Campbell[2] suggests intra-articular fusion. In our hands, the use of a bone graft from the tibia of the same side has been employed to stabilize the distal tibiofibular joint. This is driven into a drill hole in the tibia and fibula above the joint, and has proved highly successful in stabilizing the joint and in overcoming the symptoms.

SUMMARY

1. Injuries to the anterior inferior tibiofibular ligament without serious fracture are frequent, although very little is to be found bearing on this subject in the literature.

2. If a considerable degree of subluxation accompanies the sprain, disability is prolonged unless adequate treatment is given.

3. Treatment of the acute form by reduction and fixation is described; while a simple operative procedure for cure of the chronic cases is proposed.

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DISCUSSION

Lloyd Noland (Fairfield, Ala.): I am afraid I have not very much to add. The subject has been covered very well indeed, and I think that Dr. Outland has given us a most interesting paper.

Diastasis of the distal tibiofibular joint must, of necessity, be associated with extensive ligamentous rupture and may occur without associated major fracture. The mechanics, I think, are open to question. Whether or not this can occur with internal rotation, I question, but most of us are inclined to associate such an occurrence with Potts' fracture in which there is fracture of the internal malleolus or rupture of the internal lateral ligament with fracture of the lower third of the fibula. We must all agree with Dr. Outland that failure to recognize and properly treat the diastasis may, and often does, result in permanent disability.

Aldredge states in a paper read before the American Medical Association about two years ago, that definite widening of at least 2 to 3 mm. takes place at the tibiofibular joint when the foot is fully dorsiflexed, and that when the tibiofibular ligaments are sectioned, the fibula separates abnormally from the tibia when on dorsiflexion the foot reaches 90 degrees. It, therefore, seems evident that long immobilization in slight plantar flexion is indicated in every case in which separation with ligament injury is suspected in that joint. Just how long one should immobilize these patients, I do not think anyone knows. You have to try them out, I suppose. If you set an arbitrary six weeks of immobilization when there are rather violent symptoms, perhaps that is all right. There are patients, of course, on whom you will have to have metallic fixation later, but I do very firmly think the matter of putting on these braces in slight plantar flexion has a great deal to do with the end result and certainly tends toward comfort of the patient.

Robert L. Rhodes (Augusta, Ga.): I had the pleasure of reading Dr. Outland's paper, a copy of which he sent me, and as though that were not

full enough, we have his pictures and his movies which just leave very little to say.

I do have a few things that I want to suggest, mainly and particularly a slight modification in the treatment from that which he has suggested.

Dr. Outland is to be congratulated for bringing so clearly and so thoroughly to our attention a lesion often overlooked and associated with persistent pain over a prolonged period of time. However, I wish to emphasize his sentence, "The exquisite tenderness sharply localized over the tibiofibular joint" as the key in diagnosis.

For many years I have treated these badly sprained ankles, even some associated with milder type fractures, with lateral moulded lightly padded plaster splints instead of the U splint. The external or lateral splint is applied first, and extends from high up on the leg under the foot to the inner side of the instep. In other words, the lateral splint comes down and under the foot, whatever position you put it in, leaving a gap in here on the medial splint rather than carrying the U all the way around. This mesial splint is held snugly and carefully moulded until it "sets." The internal or mesial splint is then applied from a corresponding position on the leg down to about one-half or one-fourth inch above the other splint, depending upon the amount of swelling. This also is held snugly and carefully moulded until it "sets." A muslin bandage or sometimes adhesive straps in addition, is firmly wrapped around the splints. I have not had quite the success in moulding out as much of the swelling as others have, so I have used sort of a delayed measure. The muslin bandage is then replaced from day to day as the swelling recedes and the gap in the two splints allows those to be brought closer together.

In about a week or ten days all swelling is gone, the joint is snugly in position and circular plaster is applied instead of the muslin bandage. Using the moulded splints as a whole for compression, tightening them from day to day, has avoided the necessity of the windows and pads as suggested by Campbell. After their removal an elastic anklet or elastic wrap bandage is worn for several weeks.

William Darrach (New York, N. Y.): I think this is a very important lesion. We have been of the opinion for a long while that it was an important lesion, whether you have malleolar or high fibular fractures with it or not, and I am delighted to hear Dr. Marble say he had not seen one and did not know it existed, because I did not think there was anything in this line he did not know about. They are not common, I will grant that, but there are plenty of them and he has seen them but he has forgotten.

Two or three points on the treatment: I believe that four weeks or six weeks is entirely too short, because I think it takes a long while for this ligament to heal and get solid, and I think they have to be off weight-bearing, with any stress and strain on it for a longer period than that. Whether it should be eight weeks or twelve weeks, I do not know. That is the conservative treatment.

That is why for a number of years, because of this long duration, we have been cutting off a little time by putting a bolt through the two malleoli, holding the thing firmly together. At first in that soft cancellous bone, we found the head on the one end of the bolt and the nut on the other end began to cut into the bone, and we started putting plates on the end. Using those, we can start the ankle motions earlier and get them up on a walking brace at the end of three or four weeks. The bolts are usually taken out after six or eight months.

RALPH G. CAROTHERS (Cincinnati, O.): I think this is terribly important because there is a lot of talk going around that all you have to do is put in a little novocaine and tell them to go on out dancing that night; and doctors are doing that sort of thing every once in a while, then some of the rest of us get this situation when it is in a bad chronic state.

If an ankle is truly sprained in the true sense of the word, just a little stretching of some ligament without a true rupture or a true breaking of bone structures, I suppose that a little dab of novocaine, or perhaps a little colored medicine on the outside would do; but when things are really ruptured inside, as Dr. Outland has pointed out, this greater care is required. Or it may be a lesion such as I worked on last winter, that is, a rupture of the astragalofibular ligaments, which gives you almost the same thing, inasmuch as when they are ruptured and you put a little novocaine in and twist the foot in inversion, you can rock that astragalus over until it is off line about 25 or 30 degrees. Either one of those lesions requires a plaster cast and a long continued treatment, and not this proposition that the coaches are trying to put over, that is, just a strap and get the fellow back in the game the next day and perhaps have him out forever.

THE BURN PROBLEM*

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THERE is even now no universal agreement regarding the treatment of minor, much less, the extensive superficial burns in spite of the fact that these lesions are as old as fire itself. Not only is there no universal agreement, but there is no agreement in this or any other country. Every surgeon is constantly confronted with new agents, or old ones in new skins, purporting to overcome the deficiencies of those substances already available. Careful, unbiased investigations can, and should be carried out to arrive at conclusions which clinicians can use to guide them in the selection of methods and agents for the treatment of the burned area.

The lack of agreement in regard to the local treatment of the burned area is similar to lack of agreement on the treatment of wounds in general. The American Red Cross Manual gives instructions that a weak tincture of iodine be used in a wound, a procedure which must result in further tissue injury. There can be little doubt that the extent of injury in a burn is further increased by certain methods of therapy.

There is, however, agreement on many of the major factors which are involved in the pathologic physiology of the burned patient and these may be used as a basis for outlining the general therapy.

It is agreed that the pain which the extensively burned patient has is an important factor in the induction, progress and the prolongation of the shock to which these patients are subjected. The burned patient should receive adequate doses of morphine. An adequate dose is one that will relieve pain and it should be remembered that the burned patient tolerates relatively large doses of morphine.

The burned patient rapidly loses a part of his plasma volume. A portion of this is lost to the exterior, but the largest part is lost into the tissues in and surrounding the burned area. It is possible that small additional amounts of plasma are lost in other portions of the

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body, perhaps through the mediation of some toxic agent absorbed from the burned area but this is relatively unimportant in the early period of therapy. It is the loss of plasma volume into and around the injured tissues which is the principal cause of shock. Shock can, in large part, be prevented by the administration of plasma, serum, human albumin or some other adequate blood substitute. Even if shock has occurred it may be overcome by restoring a depleted blood volume with substances which tend to remain in the circulation, provided the shock has not existed so long as to make the state irreversible. It is best, therefore, to initiate the use of blood substitutes early, before shock in the traditional sense has become manifest. The cause for irreversibility in the shock reaction is even now not clearly understood. To wait for a low blood pressure and a failing circulation before initiating therapy designed to maintain a nearly normal plasma volume is to court disaster.

The use of blood during this early period may be harmful, for plasma continues to be lost while the red cells remain in the circulation. This leads to homoconcentration and an increased viscosity of the blood, to a deficient oxygen carrier, greater work on the heart, and thus to an intensification of the shock. While whole blood is useful when anemia occurs it should *not* be used in the first few days of treatment unless a *severe anemia* is present. In burns associated with other wounds transfusion may be necessary at any period of therapy.

It is now generally agreed that the administration of large amounts of sodium chloride in hypoproteinemia intensifies the edema which occurs at any given level of serum protein. The patient with an extensive superficial burn rapidly becomes acutely hypoproteinemic if he receives intravenously large amounts of sodium chloride, because a further loss of plasma protein from the vascular system occurs as the sodium chloride leaves the vessels, carrying with it a variable fraction of the plasma protein. It is because of this that it has been suggested by the Burn Conference of the National Research Council that the patients receive no more sodium chloride intravenously than they have received plasma, or a comparable blood substitute, in the same twenty-four hours. Local therapy is valueless unless sufficient plasma, serum, albumin, or a comparable substitute is utilized to maintain a plasma volume sufficient to permit the continuance of a nearly normal cardiovascular function, a normal, or nearly normal fluid exchange between the tissue fluids and the blood, and normal respiratory and cerebral functions.

The local treatment of the burned area is the most debated of all aspects of the "burn problem." Every conceivable form of local application has been recommended and applied. Complete sight has been lost of the undeniable fact that a burn is a wound, and that the fundamental principles governing wound treatment should, wherever possible, be used in the local treatment of the area of the burn.

While initially the area may be sterile, it is readily susceptible to every type of contamination to which wounds in general are exposed. It is for this reason that these wounds so frequently become infected. They are not protected adequately immediately following the injury, and they are too frequently not protected adequately during the emergency and definitive treatment. This factor must be continually stressed in the training of house officers.

The débridement of the badly devitalized tissues must be done with the greatest care lest living tissue be further injured, and with the healthiest respect for asepsis. Here indeed is an area which provides a perfect culture medium. The specific local therapy should be designed to discourage bacterial growth and to prevent further contamination. I am sure that extensive débridement under local anesthesia is rarely necessary. It is far better to leave behind small areas of nonviable epidermis and dermis than to remove viable areas from which regeneration must take place. The extensive scrubbing procedures without doubt cause more shock than the good which they do. The removal of loose skin and the gentle washing of the surface with gauze, good white soap and plenty of sterile salt solution is about all that is required. The addition of one of the sulfonamides to the material which is to be applied locally should serve further to reduce the incidence of suppuration.

Since a portion of the plasma which is lost from the circulation is lost from the surface of the burn, the local therapy should be designed to minimize this. The local tanning of the area provides a satisfactory method for attaining this objective. It is equally important, however, that the tanning agent does not simultaneously injure remaining islets of epithelium from which repair can take place. Not only must these residual islands not be injured, but repair must not be retarded by the locally applied agent. There can be little doubt but that tannic acid and gentian violet or the other dyes do, to some extent, inhibit cellular repair and perhaps cause some further necrosis. Whether they actually cause the extensive necrosis of otherwise viable tissue which has been ascribed to them cannot now definitely be answered, but I do not believe that they cause this necrosis except

perhaps when used with relatively high concentrations of silver nitrate.

In spite of the objections it should be recognized that the tanned area, as a rule, requires minimal care and permits of rapid evacuation, considerations of great importance in military surgery. If one considers further that patients so treated do not require as large amounts of plasma as comparable cases which are not tanned, it becomes necessary carefully to weigh the advantages of tanning against its disadvantages.

The pressure dressing method, which has within recent years gained a number of advocates, has a great deal to commend it. With this method an ointment containing a bacteriostatic agent, such as one of the sulfonamides, can be placed on the burned area following débridement and the part firmly supported and bound. Plasma loss is decreased by this method, the patients without doubt remain comfortable, subsequent tissue injury is minimized, and subsequent dressings kept at a minimum.

Whether this method will prove to be useful to our armed forces as it is now proving to be in the care of burns in civil life remains to be determined. It has much to commend it, for it treats the burned area as a wound. We shall have to keep our minds open in regard to this method of therapy. Surgeons in civilian hospitals should adopt it for certain patients so that we may learn further of its advantages and disadvantages. For the face, hands and genitalia it will, without doubt, be helpful, for the use of tanning solutions on these areas has led to much needless deformity. It is time that we generally agree to condemn this practice.

While the local and perhaps oral sulfonamide therapy will reduce the incidence of infection, these substances cannot be expected to correct gross errors in surgical care. If an adequate amount of a potent sulfonamide is used locally, I doubt the necessity of oral therapy. The burned patient frequently has a low renal output and great care should be exercised when those substances are used which tend to crystallize out in the urinary tract. Failure to heed this warning will lead to anuria from mechanical ostruction.

When infection occurs the local area must be treated as one would treat any other infected wound, by wide open drainage, thorough irrigation, and the local and oral use of bacteriostatic agents. To permit infection to gain firm foothold is to jeopardize the life of the patient, to prolong convalescence or to increase the resulting deformity.

It is important that the area of a third degree burn be skin grafted as soon as possible. This will reduce the incidence of infection, for such areas invariably become infected if not grafted. It will minimize deformity and will provide a healthier area than is obtained when epithelial proliferation from a few residual islands and from the wound edges is depended upon to cover the area.

The modern development of technics for obtaining grafts of varying thickness and extent for covering such areas should do away with the unsightly "pinch grafts" which are even now too frequently used for covering the denuded area. Such plastic procedures require some additional knowledge, but the surgeon who does not possess this should not attempt the reconstructive stage of therapy.

The intelligent care of the burned patient requires an intimate knowledge of basic surgical principles and a working knowledge of surgical physiology. While the intern may possess the latter, he does not have the former. These patients are often relegated to the youngest house officer for care when in reality they deserve the attention of the most experienced member of the staff.

It is highly likely that burns will constitute a considerable group of the casualities in our armed forces as well as in civilian life during the next few years. We must meet the problems which they present without bias and prejudice.

DISCUSSION

WILLIAM DARRACH (New York, N. Y.): I hope I never have to be in the position of living on the gleanings of a field that Ravdin has harvested, because he leaves very little for the gleaner.

There are one or two things that I would like to emphasize, however, and that is, I think with this problem of burns, the thing which he brought out, and on which I would like to place the greatest emphasis, is that burns are wounds and should be treated on the same fundamental principles that wounds are treated.

Secondly, I think that we can divide our present day knowledge into the things we know and the things we do not know. Of the things we know, I think first comes what he said about pain. Pain must be relieved because it makes the patient sicker if it is not, and not only pain at the time but pain during the ensuing days and weeks.

The second thing I think has been pretty well cleared up and that is the question of the loss of the blood constituents. Most of these patients do not need red cells, most of these patients do not need salt; and if we think of what we heard this morning about the autopsies on the badly burned patients, the kidneys and the kidney tubules, we will not give them too

much salt. But what they do need is plasma, and plenty of plasma. I think that is pretty clear and definite.

I think another thing on which we all agree is that one of our most important duties is to prevent infection, and to prevent infection by means which will do good and not do harm, that is, by means which will get rid of the contamination with as little damage to tissue as possible.

What he said about gentle washing with a soap solution instead of violent scrubbing is along the same lines. He spoke of the use of strong chemicals to get rid of bugs which at the same time kill body cells. I think we are all in agreement on that.

There is another fundamental principle in the treatment of wounds that we all agree on: they all need rest, and that means general rest and local rest.

I think one of the things we are not sure about is what to do with the dead tissue. We realize that in compound fractures and in contaminated wounds, the object is to get rid of the devitalized tissue and the foreign material. How far shall we apply that in burns? I think we do not know to what extent we should débride and get rid of the dead tissue. That is why the tannic acid treatment which seems clinically to have helped so tremendously in the ways in which it has been used, perhaps is theoretically wrong. I have not been able to think that thing through; I do not know. Here we are covering this wound, or continuing the covering of the wound by tissue which is practically dead. Perhaps that is wise; perhaps it is not.

On the question of coverage, I think clinically everybody agrees that we must get these wounds covered in some way to cut down the plasma loss, to cut down the pain, and to hasten healing and quiet down the edema. What is the ideal coverage? I cannot help but believe that theoretically, at least, the ideal coverage is skin. Now if means can be worked out whereby skin grafts shall be done at the earliest possible moment instead of later on, I think we will make advances in this treatment. It has been done in some places but I do not think we know yet how early it can be done safely.

As for the sulfonamide drugs, I want to put in one word about those and I like to say it as often as I can in regard to their use in wounds, whether compound fractures or burns or anything else: Let us remember that they are aids and not substitutes for good surgery.

JOHN F. GILE (Hanover, N. Y.): There is very little that I can add to this discussion after Dr. Farmer's paper, Dr. Ravdin's paper and Dr. Darrach's discussion. About the only thing I can do is to agree with everything that has been said. I do not think there is any question about everybody's agreeing with the first-aid treatment, getting these people warm, and the use of morphine and plasma, and so forth.

As far as what you are going to put on locally, I think there is still, of course, considerable discussion. We believe that in patients who look as though they were going to have considerable dehydration, tannic acid is the

best treatment. On the other hand, as has been emphasized here, we believe that it should not be used on the extremities, and so forth. We have people coming in, upon whom the treatment has been decided on the outside by the first-aiders with a very generous use of tannic acid. We think that perhaps somewhere in the Red Cross book, that might be changed somewhat. We have had cases come in, in which we wouldn't primarily pick tannic acid as the treatment, on fingers and so on, on which it has been put, and we are then pretty well sold as to what the method of treatment must be.

We believe that steariform and wet saline solution with pressure is about as good as anything we have; and we are of the opinion that patients get along and look about as well as with any other type of treatment. That is what we are using with pressure dressing, particularly, and splinting at times. We have also recently in two or three cases on the lower extremities—and I see no reason why it couldn't be used on the upper extremities—put on the splints, so you get the legs up; and where there are bad burns on the back of the leg, you get it off the bed and up so you can dress it, and it is certainly more comfortable.

We have used no sulfa drugs in oil. We have used the powder on some of these burns and believe that it cleans them fairly well. But I think one other word of warning should be given. You should not continue to use it too long, as we believe that after a certain time it inhibits new growth. Whether that is absolutely true or not, I do not know, but we have rather believed so. We agree absolutely that early skin grafting is very important, getting them grafted as soon as possible, and starting motion, and so forth, of the part as soon as possible.

Douglas Ackman (Montreal, Canada): I should like to describe the method we are using at the General Hospital in Montreal. Before going into details, I might give you a little preview of how it occurred. Because of the emergency conditions and the fact that we are surrounded by munitions plants in Montreal, and the remote possibility of air raids, it was considered necessary to organize some kind of plan. In doing this, we had a committee which operated, and we have achieved a certain working plan which, with the help of a few slides, I would like to show you now.

We have one chart which gives the timing in the treatment. The timing object presupposes the development of the treatment as the burn itself progresses. We have to have two teams operating at the same time, one a shock team on a twenty-four-hour basis, and the other a burn team for local treatment, both under the supervision of a senior surgeon.

This chart also outlines laboratory examinations and clinical data. We have divided our burn timing into a period of shock—the first forty-eight hours; the period of toxemia—the second to the fifth day; infection and granulation between the fifth and fourteenth day; and finally, a healing time which we put from the second to the sixth week. Our object is, first, to

treat the patient's shock before attempting to undertake local treatment, and the treatment is shown on this chart.

Just one word about the treatment with plasma. It is controlled by repeated hemoglobin estimations. We have to have a hemoglobin estimation done every hour for the first four hours, and thereafter every four hours, especially in severe cases. In the second hour, if the case is desperate, we give cortin, 25 cc. intravenously in extreme cases, and continue that cortin 10 cc. intramuscularly as indicated. Plasma is given according to a plan.

In the local treatment which is divided into two parts, one sees the employment of an eschar treatment for trunk, thigh, legs, feet, arms and forearms, and on all critical areas we are using a sulfathiazole emulsion which has been developed at the General Hospital.

The redressings that are done are also shown on this chart. In the second twenty-four hours, it is sometimes necessary to redress our critical areas, and at other times it is not. It depends upon the serum loss and the appearance of the dressings. During the second twenty-four hours we have a complete blood chemistry done. In the second period of toxemia, the sulfonamides are introduced if they are needed, blood substitutes as indicated, and if in the eschar we have infection, it is unroofed and the emulsion dressing applied. All critical areas continue to be redressed at suitable periods.

In the period of infection or granulation, as the case may be, we carry out the treatment according to plan. If anemia develops, transfusions are used, and diet and vitamins as indicated. Again if infection occurs, it is unroofed and the emulsion dressing applied.

We have believed it is better, no matter what type of eschar is used, that it be removed if necessary by surgical measures at the end of the second week, and thereafter if it is a second degree burn, it is treated by emulsion dressings. If it is a third degree burn and requires skin grafting, this is done after the use of saline dressings until the granulations are suitable.

A second chart, which we are using, is to control the treatment. The nurses in attendance on the ward have these charts in front of them; they do them, themselves, and they have the basic chart to go by. One column indicates blood pressure, and another the pulse reading. Hemoglobin estimations are carried out in these cases. There are one-hour periods, and consequently everything is done on a systematic basis. For notations regarding treatment, and for purposes of laboratory examination, we use other indications on the chart. On the back of the chart an estimate of the burns is given.

The emulsion which we have been using at Montreal General Hospital is an oil and water emulsion with some microcrystals. The technic we are employing is as follows: We clean the wound under anesthesia, preferably, using morphine only, and lavage it with saline solution, spray on some powder as a frosting, and then use the emulsion in single thickness strips of

gauze, either ordinary hospital gauze, or better, a curtain mesh, which we call a sulfa mesh. These can be made beforehand or made up as you dress. We apply the dressings over these and use some padding. We have had no experience with pressure dressings but we do use plaster moulds.

CHARLES LUND (Boston, Mass.): I first want to thank the society for the invitation that I have had to attend the meetings, and to say how much I have enjoyed them. I do not want to prolong this discussion too long, but I have had a recent experience that I think is of some interest. In trying to treat the class of burns that is now essentially insurable, we run into new problems when we use these new methods. Two weeks ago I was confronted by a man burned by flaming alcohol which got onto his cotton shirt and burned him completely front and back, from the waistline of his trousers up.

The face and the hair were only first degree. About a tenth of that area, on the chest, was a third degree burn and the rest was a bad second degree burn, and there was also a circular second degree burn of the right thigh, and a small patch on the left thigh. We calculated about 70 per cent of the body was burned. The genitalia were not burned and the legs below the knees were not burned.

Recently, a patient died in Boston where the medical examiner believed that compression of the chest from a circular eschar was a major contributing part of the cause of death. I knew about that case and because I was impressed by some things I learned at a meeting in Cincinnati, I tried to use boric ointment with pressure. You cannot put on much pressure when you are dressing with boric ointment completely around the chest.

The wound was cleaned, shock was treated during the cleaning, and then enormous dressings were put on. There was tremendous leakage of plasma for the first two days, and during that time, 2,000 cc., twenty units of 25 per cent albumin were given, diluted in 10,000 cc. of saline. That brought his hemoglobin, which was 128 two and a half hours after injury, down to 100. Following this, he was given saline, his hemoglobin sank to 80, and his urine output went way up to 3,000 and 4,000 cc. a day. He was given sulfadiazine by mouth and he remained in what looked like good condition.

On the fifth morning he began to breath a little heavily and I did not like his looks. I thought he might be getting pneumonia. There was no way to examine him for pneumonia on account of the burn. I consulted with an excellent pneumonia man, and we did not do anything very active. Later that day a plasma protein was taken for determination, and at 9 P.M. he was dead. He died with definite respiratory signs the last two or three hours. The plasma protein came back the next day, 3.2 albumin and 1 per cent globulin, a total of 4.2 in a laboratory where the normal is around 7.

That man should either have had blood plasma or whole blood during the last forty-eight hours of his life, and we thought because his urine output stayed up so well, he looked so well and his hemoglobin was so high, that he did not need it.

I think we will find that this additional test is important and must be repeated frequently and reported promptly. This perfectly splendid chart covering everything might possibly suggest that you do a little more work and get plasma proteins in.

I think that is about the biggest dose of human albumin that has been given. That dose, incidentally, is the product of forty pints of blood.

FREDERIC W. BANCROFT (New York, N. Y.): I have some cases* showing a fatality which I think are worth while reviewing now:

A child with a burn of two days' duration came into the Lincoln Hospital and was débrided. In the upper end there was one little area of epithelium. There was edema in the subcutaneous tissues and down to the large vessels in the back. This shows very definitely, I think, the amount of edema that occurs in the subcutaneous tissues in the vicinity of a burn, and where we could save a lot of our plasma loss and prevent edema from occurring.

Another case is that of a six-day burn in which infection occurred rapidly. At the upper end, where the burn was, nothing but a parchment membrane, a little dark line, appeared. That was the separation between the living and the dead tissue. This must be débrided and treated.

We have also seen one of the rare cases of ideal treatment of the eschar, showing the eschar up above, the correct location of the sweat glands no infection beneath the membrane and a firm splint of the membrane. I think that not only is an eschar of advantage, but if we débride that eschar, we must handle the tissues gently in order to preserve the layer of epithelium.

GROVER C. PENBERTHY (Detroit, Mich.): I do not want to prolong this discussion, but the treatment of burns is a timely subject and Dr. Ravdin has emphasized the importance of the surgical care. Dr. Darrach reémphasized it, as have others, so I would like to speak for a moment and refer to the work that Dr. Johnston is doing in Detroit for the National Research Council.

This morning we discussed the frostbite of the mariner. Now we may have the same problem to deal with in the case of the mariner burned with oil. A very thick engine oil had been applied to the burn. That burn with oil is treated with ordinary lard and an emulsion formed, and after that emulsion has been completed, the wound is thoroughly washed with soap and water and irrigated with saline or boric acid. This prepares that wound for the coagulant, whether you use gentian violet or tannic acid.

Dr. Darrach said he wondered what should be done in the way of débridement. I think it should be the necrotic treatment, the blisters opened, and so forth.

Dr. Kreuscher asked the question whether the emulsion was applied as a dressing and I can answer no, just a cleaner. The President referred to the Pearl Harbor situation. There they had many oil burns to deal with.

* Dr. Bancroft presented illustrations of these cases.

E. Payne Palmer (Phoenix, Ariz.): The first problem in the severely burned patient is the treatment of shock. Many of these patients can be saved from sudden death and moved to the hospital by the standard shock and burn treatment—these fifty-seven varieties we have of burn treatments —if they are treated for shock at the site of the accident and not moved until the condition is improved. Too many of these patients are moved while they are still in the state of severe shock, and the mere fact of turning them or attempting to move them may terminate life right there, or the transportation to the hospital may terminate life.

In these various demonstrations here, you give them morphine, which is all right; you give them the cortical extract; but you must give something that is going to get the patient out of shock as quickly as possible. As I said this morning, and I am going back to it again, give big doses of coramine to get the patient out of that immediate shock, and when you get him out of that period, you can give the coramine intramuscularly in order to get him into the hospital. It is only a temporary affair, but you must have something which you can give these patients until they get into the hospital to have the standard treatment which you are going to give them there.

But do not move these patients in their state of shock until their condition will permit them to get into the hospital, until their muscle tone is improved, until their venous tone has improved, and until the flow of blood in the veins has improved.

Hubley R. Owen (Philadelphia, Pa.): I will not prolong the discussion any longer except to say that from 1937 to 1940 on our service in Philadelphia General Hospital we had the opportunity of treating firemen for extensive burns. We tried all methods and the method we used more extensively than any other was the one mentioned by Dr. Wagner, the ambrine method or paraffin, and tannating these wounds. But I think the important thing we have gathered from this is to treat the patient first and the burn after.

I cannot come around to using engine oil on these burns. It does not sound like good surgery, any more than using Carron oil, because it must be dirty, you have a primary wound to deal with and you will get infection if you use Carron oil or engine oil. I do not like the sound of that.

THE TRANSPORTATION AND OPEN TREATMENT OF SEVERE FRACTURES

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IN the first World War, we learned much about the transportation of patients with severe fractures. During the first two years, all kinds of splints were used: metallic, wood, and plaster of paris. Many times severe infections, hidden abscesses under plaster dressings, atrophied muscles, and gas gangrene developed which necessitated amputation.

The proper first aid and transportation of a patient with a fracture has a marked influence on the end result. Love's statistics of the World War show that more than 30 per cent of all war casualties admitted to the evacuation hospitals were compound fractures. The British, during the first two years of the war, had a mortality of 50 per cent in compound fractures of the femur. They used principally the Liston wood splint. After discarding this type of splint and adopting the Thomas traction splint, their mortality rate dropped to 15 per cent.

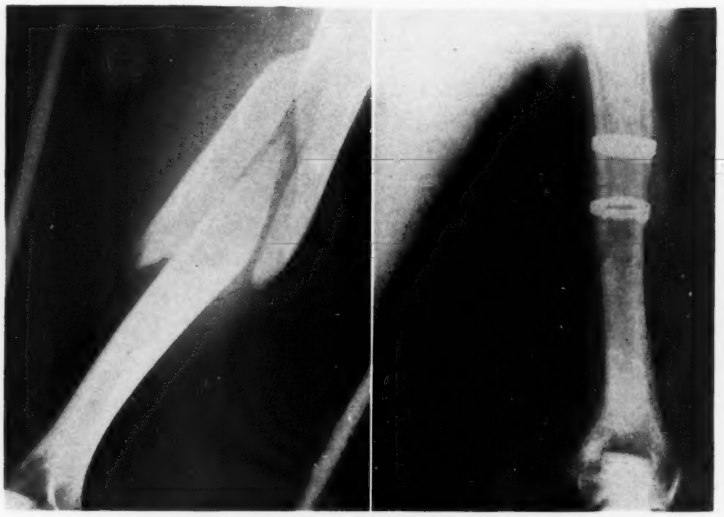
The Allied Medical Staff adopted the Thomas, Murray-Jones, and Kellar-Blake splints. They were put into service and proved very practical and efficient. They were made of iron and were light, unbreakable, and hundreds of them could be placed on a truck and be delivered to our Field hospitals. Their application to an extremity was very simple. It is said that every soldier in the British Army was taught how to apply a Thomas splint before he was sent into the firing line. Our Army and Navy are using these same splints in the present war.

ADVANTAGES OF TRACTION AND SUSPENSION IN TRANSPORTATION

The advantages of traction and suspension in transportation are: (1) The lack of pain; (2) ease in handling and transportation; (3) accessibility to wounds for treatment; and (4) no damage to muscles, nerves, or blood vessels during transportation.

From personal experience during the War and also since that time, I am convinced that these are the best first-aid and transportation splints available at the present time.

Every case of fractured extremity that came to our hospital during the last year of the World War had a Thomas, Murray-Jones, or a Kellar-Blake splint applied, and in many instances the treatment was continued in the original splint.

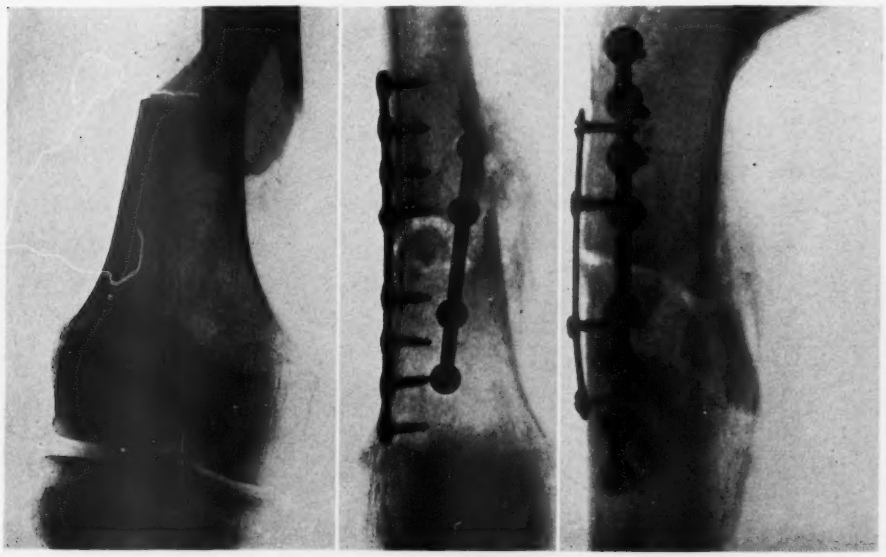


A B

FIG. 1. Mr. A. O., age fifty-four, slipped on the ice; comminuted fracture of the right humerus; two gold Parham bands applied. The patient made an uneventful recovery. Bands still in place fourteen years later.

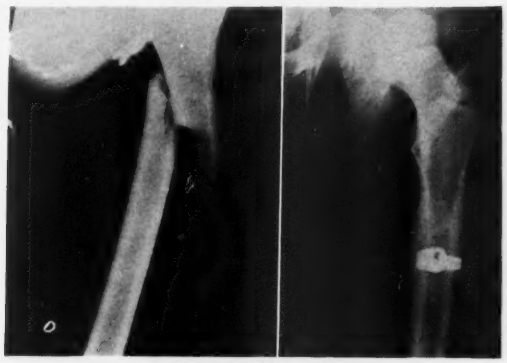
On my service at the American Red Cross Military Hospital No. 3 in Paris, 72 per cent of all cases were compound infected fractures, and all of them were treated by débridement, Dakanization, traction and suspension in one of the above splints. In the present war, sulfonamide therapy will be used: sulfanilamide in the wound and sulfathiazole by mouth to combat infection. The first experience of this treatment on a large scale by our American surgeons was at Pearl Harbor where all compound fractures were treated by débridement, sulfanilamide in the wound, sulfathiazole by mouth, and the extremity placed in a Thomas or Murray-Jones traction splint. All the surgical teams were impressed by the results of the sulfonamide therapy. Drug reactions were rare. No tetanus developed as tetanus toxide had been given on entrance to service. Several cases of gas gangrene developed in which wounds had been closed by suture (which was contrary to Army instructions). These patients were treated by débridement, sulfonamide therapy, and *x*-ray. No amputations were necessary. Dr. Moorhead, who was in charge of one of the surgical teams, says, "The splendid results obtained at

Pearl Harbor depended on (1) Early treatment within 6 hours; (2) team work; (3) available Plasma for shock treatment; (4) débridement—no suturing of wounds; (5) sulfonamide therapy."



A B C

FIG. 2. Fracture at the junction of the middle and lower third of the femur. Tongs were applied, then two Lane plates.



A B

FIG. 3. A, fracture of the upper third of the femur of a child four and one-half years old. Parham band applied; B, four years later.

The open reduction of severe fractures is now more generally used than the closed method. This is evident to anyone attending the larger clinics. The reasons for the increase are: (1) We are treating more severe fractures than we did twenty-five years ago. (2) Every severe fracture is a potential liability to the surgeon in charge. (3)

With the sulfonamide therapy, i.e., sulfanilamide in the wound and sulfathiazole by mouth to combat infection together with the improved instruments, plates, screws, and technic our results are better.

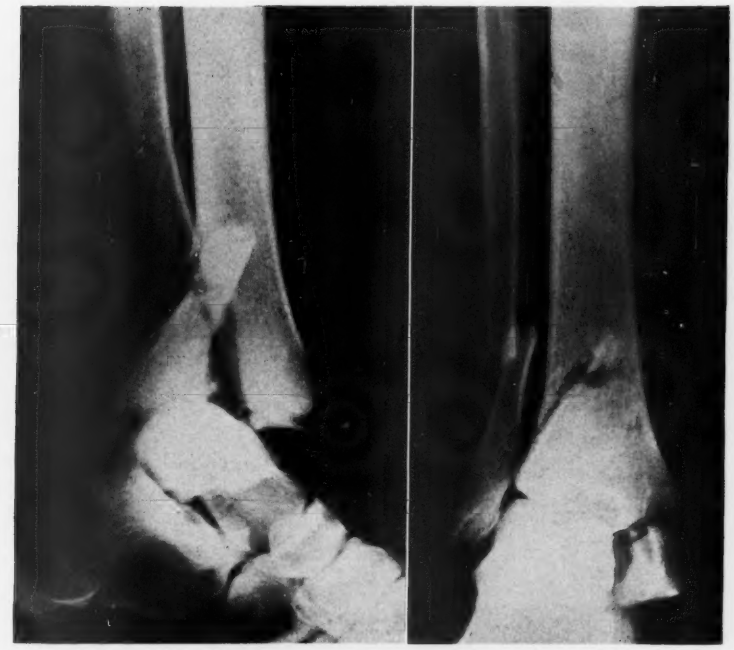
A B

FIG. 4. E. B. B., age thirty-five years, fell a distance of forty feet from a bridge on May 16, 1926; causing an oblique fracture of the upper third of left femur. Open reduction—Parham band applied. leg in Hodgin's splint; uneventful recovery. Band still in place sixteen years later.

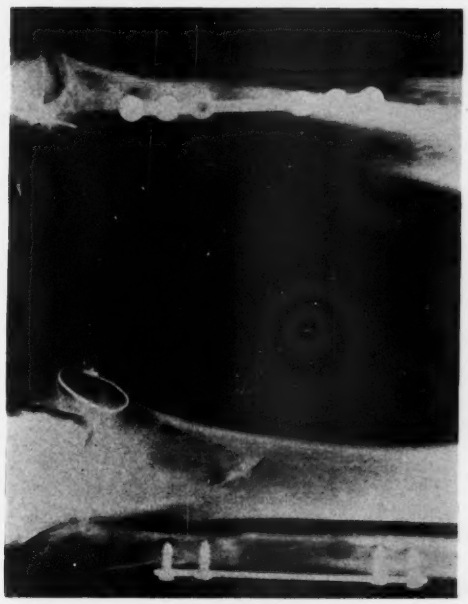
It is interesting to review some of the older textbooks on the treatment of fractures which bring out this gradual change from the closed to the open method. Scudder's Sixth Edition, published in 1907, mentions only three cases treated by the open method; one a fracture of the olecranon, another a fractured patella, and the third a fracture of the surgical neck of the humerus. A silver wire was used in all of these cases and no mention or illustration of a Parham band or a Lane plate is found in this edition. In his Eleventh Edition there is page after page on open reductions and illustrations of the Parham band, plates of various kinds, screws, pins, etc.

My first ten years of practice were spent in a mining district. At that time we treated all of our patients with severe fractures by the closed method, using either molded plaster splints or plaster casts.

During the World War we treated our patients with severe fractures in traction splints. Shortly after my return from war service,



A B



C

FIG. 5. J. K., age twenty-five, fell from the top of a box car on October 20, 1936, causing comminuted fracture of the distal end of the tibia and fibula of the right leg with forward dislocation of tibial fragment on astragalus and a linear fracture distal end of left tibia with no displacement. Open reduction right leg using Lane plate and aluminum bronze wire. Discharged from hospital December 23, 1936. Plate and wire were removed.

I became associated with Dr. A. F. Jonas, then Chief Surgeon of the Union Pacific Railroad, who taught that a good anatomical result was necessary in order to obtain a good functional result. We all

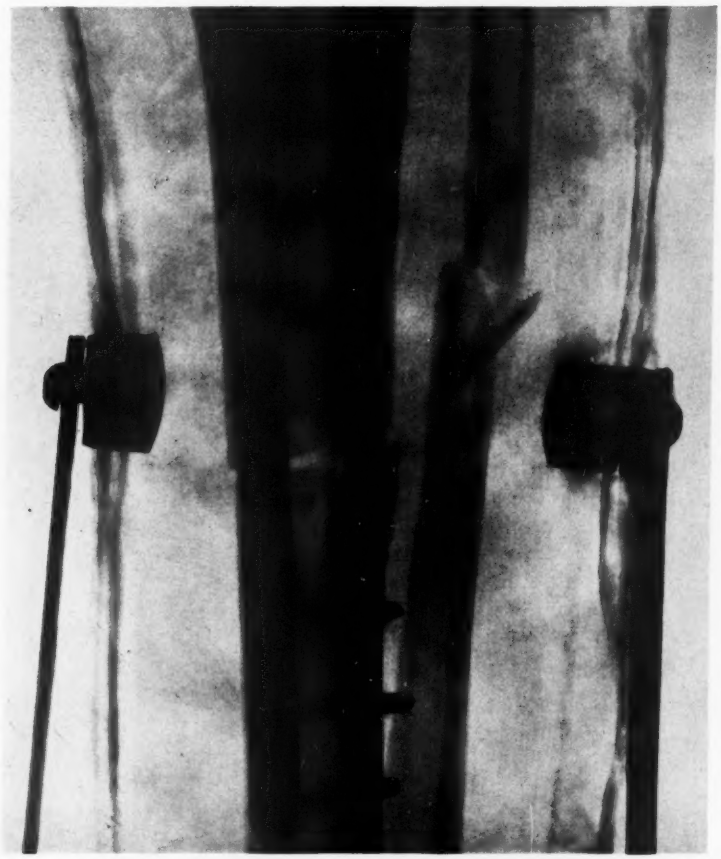


FIG. 6. Mr. O. K., age fifty-nine, July 26, 1942, compound comminuted fractures of tibia and fibula of right leg, junction of upper and middle third. Open reduction July 28. Stainless steel plate with screws through both cortices (as advocated by Dr. Clay Murray). A Thomas splint with traction was applied. August 15, the wound was healed, and the leg was placed in a plaster cast extending from toes to mid-thigh, with incorporated walking iron. He was dismissed from the hospital August 16, walking with crutches.

know that the two main objectives in treating any fracture are to prevent deformity and to restore normal function. Dr. Jonas did many open reductions using Parham bands, Lane and Sherman plates, and aluminum bronze wire.

A few years ago the British Medical Association appointed a committee to investigate relative results in fractures, open and

closed methods. Their conclusions were as follows: (1) Nonoperative in children 90.5 per cent good functional results, operative in children 93.6 per cent; (2) progressive depreciation of functional results from nonoperative as age advances; (3) operative treatments give higher percentage of good results at all ages; (4) the best way to get a good functional result is to secure a good anatomic result; (5) a method that does not definitely promise a good anatomic result should not be chosen; (6) for best results operate early, not after failure of nonoperative treatment; (7) operative treatment requires skill and expense; (8) the mortality rate is practically nil.

In industrial work such as I was engaged in, it is absolutely essential that our reductions are as nearly perfect anatomically as possible; because if there is any liability in connection with an injury and an unscrupulous lawyer would show an *x*-ray film in which the fragments of a fracture were not in perfect alignment, it would make a most damaging Exhibit A against us.

At the January, 1941, meeting of the Committee on Fractures and Other Trauma of the American College of Surgeons at New Orleans, the open reduction of fractures was discussed by Dr. Murray, of New York, Dr. Venable, of San Antonio, Dr. Berg, of Pittsburgh, Dr. Morrison, of Boston, and Dr. Speed, of Chicago. They expressed their views in regard to the most satisfactory metallic plates and screws to be used in these open reductions. Dr. Venable reported the results of his research work showing the influence of the tissue fluids on the various metallic plates used at the present time, which were as follows: (1) The microammeter shows that Vanadium steel produces 200 milliamperes; (2) stainless steel produce 50 to 100 milliamperes; (3) stainless steel passified by HNO_3 produces 10 to 20 milliamperes; and (4) vitallium produces 1 to 2 milliamperes momentarily then 0.

The amount of electrolytic disintegration of metals and consequent erosion of bone seem to be related to the current flow recorded by the microammeter. A metal that will produce many milliamperes will produce much erosion of bone and the metal itself will disintegrate. A positive proof of electrolytic action in the body is the ability to recover constituent metals of an alloy from the tissues adjacent to it.

Dr. T. A. Carnes, of the Republic Steel Corporation, has shown by *x*-rays that vitallium plates and screws, which are made of cast metal, contain air bubbles and are more easily broken because of these defects. The Republic Steel Corporation in their research

laboratories are working on different combinations of elements for plates which promise to be more ideal for fracture work than any plates on the market at the present time. All metallic plates, now available, show some electrolytic action between the plates and the body fluids, some to a greater extent than others.

The Sherman Vanadium steel plates, according to some authorities, corrode; sinuses form and the plates have to be removed. I understand, however, that molybdenum has been added to the metallic constituents of the Sherman plates and a new stainless steel plate is the result which is subject to very little, if any, corrosion by the body fluids.

During the past twenty-five years, I have used all kinds of metallic plates, screws, Parham gold bands, and silver and aluminum bronze wire in my fracture work, and the only cases in which I have had to remove the plates were in those in which sinuses had formed. These sinuses I attributed to low grade infection, loose screws, or improper fixation. Since the report made by Dr. Venable on the electrolytic action of the body fluids on the plates and screws, I believe that perhaps the occasional necessity of removal of a plate may have been due to this cause. Very few removals, however, have shown any evidence of corrosion.

Dr. Berg in his twenty-nine years of experience with the Sherman plates, states that he believes it is the technic of application of the plate rather than the kind of plate used.

In conclusion, I will state that in severe fractures we will have fewer permanent deformities, better functional results, and the period of disability will be shorter when an open reduction has been performed. The success of the internal fixation of fractures will depend upon: (1) The careful preoperative preparation of the patient, i.e., the field to be operated upon should be cleaned and tincture of merthiolate applied twenty-four to forty-eight hours before the operation. (2) Preoperative preparation of splints, open plaster casts, molded plaster splints, or splints such as Thomas, Kellar-Blake, or Murray-Jones should be available where traction or suspension is to be used. (3) Spinal anesthesia is the anesthetic of choice in fractures of the lower extremities. (4) Rigid fixation is secured by the use of a chromium nickel steel plate with screws extending through both cortices. (5) The end result will depend upon the experience, intelligence, and good judgment of the surgeon in charge.

TREATMENT OF MINOR CASUALTIES

WILLIAM DARRACH, M.D.

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NEW YORK, NEW YORK

MANY interesting laboratory studies and clinical experiments are being carried out as to the best method of treatment of burns, compound fractures and shock. The reports from Spain, England, Germany and Pearl Harbor show that distinct advances have been made since the last war in the handling of major casualties. To those of us who remember our sad efforts to combat shock in the last war it is a great joy to know that there are already available to the armed forces 150,000 packages of dried plasma with the means of administering it under the crudest conditions. This number before long will reach 600,000. The effects of the sulfa drugs on the outcome of abdominal wounds and compound fractures is another source of encouragement.

We hear little, however, about the handling of a group of casualties which for two reasons should receive more attention. I refer to the so-called "lightly wounded" or "minor casualties." They deserve more thought because they not only comprise the majority of all injuries but they are the only ones who have much chance of getting back into service. The figures of the last war show that 53 per cent of the casualties were soft part wounds of the extremities without fracture or injury to the large vessels or nerves. Added to these were many similar wounds of the head, chest, back and buttocks. From the standpoint of numbers they deserve primary consideration.

Of all the penetrating wound of the head, chest, abdomen, spinal cord and of the compound fractures, the number who ever returned to active duty is very, very small. If one of the main duties of the Medical Corps is to maintain the strength of the fighting forces, it seems rather silly to focus attention on the group who are permanently out of the picture.

Though we may differ on some details of wound treatment, we will all agree that the time between the trauma and the operative treatment is one of the most important factors in determining the period of disability and the successful result. This holds true of

the minor wounds as well as the major ones. Yet we have all seen long queues of lightly wounded, waiting and then passed farther and farther back while the operating rooms were filled with patients with serious wounds of the chests, abdomens and compound fractures and a valiant but usually unsuccessful attempt made to restore them to duty.

We all accept the wisdom of débridement for compound fractures and penetrating wounds of the thorax and abdomen and yet forget that it originally was proved of value on minor wounds. If a small superficial wound be débrided early and sutured, the disability period may be measured in days. If it is neglected until infection is well established, the victim may be incapacitated for months; yet these patients rarely get anything but a dab of iodine and a change of dressing during their long period of travel.

If the problem were faced in cold blood and courageously, the methods would be exactly reversed. These lightly wounded should have the right of way and the time and attention of the best surgeons at the first operating station they reached. After they had been attended to, the serious cases should have their chance. However, the numerical strength of the fighting forces is not the only responsibility of the Medical Corps. Morale must also be maintained. If the soldiers learned that the seriously wounded were being put to one side, if the people at home learned that their boys were being neglected, there would be trouble brewing. My plea is not that such a complete reversal be made, but that plans should be made in each advanced hospital for the prompt surgical care of both types.

The handling of the minor casualties differs in several ways from that of the major cases. In the first place they rarely need preliminary shock treatment. Secondly, the operative procedure is much simpler. In both groups the stay in the operating room may be divided into three periods: (1) the preparation, (2) the operation itself and (3) the application of dressings, including splints. In the major cases, the second or operative period occupies a majority of the total time, perhaps 70 per cent. In the minor cases this middle or operative period may be no longer than the other two. Experience in the last war seemed to show that operating teams handling the major cases would do well if they averaged one case an hour. With proper organization it was demonstrated in several places that one operating surgeon could handle six to eight minor cases an hour. In most operating hospitals the usual practice was to have one table for each team and all three stages were done by the operator and his assist-

ants. Sometimes, by using two tables, with an extra Corps man, the first and third periods of preparation and splintage could be made to overlap the actual operating. In a few places by assigning extra assistants and three tables to one operator, the minor cases were put through at a much more rapid rate. If intravenous anesthesia were used, it would be possible to have the preliminary work done by a well trained Corps man or nurse. The same holds true of the third period or application of dressings. That is, the Ford assembly line principle is applied to this group. If inhalation anesthesia is necessary, a second anesthetist would be required.

The after treatment of the minor cases requires far less nursing and a considerable proportion of them would become ambulatory within a few hours or days. This would mean adapting ward service and arrangements to the different groups. Different plans for the convalescent period should be made according to the conditions under which the total hospitalization scheme is working. My understanding of the present Army plan is to have a surgical hospital assigned as far forward as possible for the so-called "non-transportable" cases. This would fulfill at least some of the humanitarian duties of the Medical Corps. The problem of restoring men to active duty will fall largely on the shoulders of the evacuation hospitals. It is hoped that in these units plans will be made to divert the stream into two separate channels to be handled as described above. Under certain conditions it might be wise to have a separate hospital assigned to each type.

This principle of giving the lightly wounded an even break can be applied in our home defense plans as well as to the problems of the armed forces. No one can foretell how much of what has happened in England will be repeated here, but plans to meet catastrophes are being made in most of the larger centers. Arrangements have been made in most hospitals for meeting a sudden demand for handling considerable numbers of injured people. These injuries may be from bombs, explosions, train wrecks or other causes. It is hoped, as plans to meet these emergencies are made, it will be provided that these lightly wounded will receive the same prompt, early operative treatment as the more serious cases. Most of the minor casualties can be handled on stretchers and do not need the more elaborate set-up of the formal operating room. It would seem wise to have separate teams assigned for the two types of work.

In meeting this problem either at the front or at home one of the most important considerations is the proper primary sorting of cases.

One of the most experienced men on the surgical staff should be stationed at the receiving center in order that he may decide to which group each case should be sent. The lightly wounded could be sent directly to the operating group appointed for this purpose. The more severely wounded, divided into three groups, would be sent either (1) directly to the shock wards, (2) directly to the major operating room, or (3) be made comfortable during their brief period of stay.

SUMMARY

Much attention is being paid to the handling of burns, compound fractures and shock and the use of the sulfa drugs for war casualties. We hear little, however, as to the treatment of the lightly wounded which not only comprise the majority of traumatic conditions in wartime but are the best possibilities for early, complete return to duty.

The time between injury and operative treatment of wounds largely determines the period of disability. One duty of the Medical Corps is to maintain the numerical strength of the fighting forces. Although very few of the seriously wounded ever get back to duty, they not only have received priority in treatment but have occupied most of the attention of surgical teams. Minor casualties should get at least an even break. With proper operating room organization, six to eight minor cases an hour can be handled as compared to one major case an hour. After-care and convalescence can be provided on a much simpler scale than for the seriously wounded. Plans to handle minor injuries should be made in hospitals at home if any catastrophe causes a sudden rush of injured people.

DISCUSSION

H. GURTH PRETTY (Montreal, Canada): I hardly think that Dr. Darrach's paper requires discussion. The author has pretty well completed the arguments pro and con. It is interesting and important and to the point.

One important feature is that he refers not only to the armed forces, with regard to the lightly wounded, but also to the civilian or munition forces in connection with the lightly wounded, and I think that point should be stressed. Most of us are inclined to let some first-aid person or nurse segregate the cases coming to the hospital, and the result is that the moderately lightly wounded or slightly injured munitions' workers are relegated to the out-patient department where a junior house officer does the work, with the result that at some later time they are admitted

to the ward with some form of infection, cellulitis or otherwise, which considerably incapacitates them and keeps them from returning to duty.

The other point that Dr. Darrach stresses is very important, and that is the question of two front-line surgical centers, one for the severely wounded and one for the lightly wounded, which is equally important, and again stressing the importance of the supervision of a trained surgeon for the segregation and operation upon these patients.

I can well recall the latter station. I was a medical sergeant and anything less severe than a compound fracture seemed to be left entirely in my hands. I had no medical knowledge and I dispensed as I saw fit, and I certainly realized that many of the men must have been incapacitated by my over or underindulgence in treatment.

Just a word on the lightly wounded from the point of view of more or less civilian and munition accidents. There is one point which I would like to stress, and that is, in the last three months, I have run into three more or less lightly wounded individuals. One was an Air Force mechanic who, on coming home for the week-end, cut the flexor longus to his thumb, not with a beer bottle but with a ginger ale bottle. This made me particularly disgusted because we are not allowed to touch any of the armed forces, although they are in Canada, and they are supposed to report to the Air Force personnel. This happened at midnight. He was not able to get in touch with any of his Air Force personnel and he finally came to me at the hospital where we repaired the tendon, kept him for a day or so, and the Air Force immediately took him in hand and reprimanded me for attempting to suture the flexor longus tendon.

The next was a munition's worker who works in a shell-filling plant, putting fulminated mercury in percussion caps. On one occasion some of these caps exploded and he was literally peppered with little particles of brass turnings and filings and bits of bronze. He was treated lightly at the plant, merely by dressings, only to find out that about three weeks later he was suffering from copper poisoning.

The third was a ballistics engineer who learned ballistics from his own point of view. During the trial of a Maxim gun, somebody pointed the gun in the wrong direction. The bullet went through two oak partitions of the office and finally struck this unfortunate individual just above the antero-superior spine on the right side and tore into the loin making an area about three to four inches in diameter. The interesting thing about that is that he learned ballistics from his own point of view, and the other thing was that they were keen enough then to see that immediate surgical procedure was carried out and they did not attempt anything further.

I think the point made here by Dr. Darrach is the strict surgical appreciation of these minor wounds, both in the armed forces and the civilian worker.

RUPTURE OF THE DIAPHRAGM DURING A POWER DIVE*

DESCRIPTION OF A NEW METHOD OF REPAIR AND CASE REPORT

FRASER B. GURD, M.D., C.M.
MONTREAL, CANADA

WE have all heard of the period of "blackout" suffered by members of the crew during power or dive bombing and rumors, at least, of the methods adopted for its control have been current. Recently, I have had under my care an aviator who presented a lesion due to power diving which, to my knowledge, is new. If rupture of the diaphragm occur at all frequently, and particularly if the pilot of the machine be the victim, a possible added explanation of crashes under these circumstances may have been discovered.

On August 12, 1941, I first saw A. K., thirty-nine years of age, who had a few days before come to Canada as an Air Force member of the Czechoslovak Military Mission. His history showed that prior to 1938 he had been a member of the Czechoslovak Civil Air Force and had been examined and re-examined medically while in this service. In 1938, he joined the Military Air Force and was again re-examined. Following the invasion of Poland he escaped from Czechoslovakia and after incredible hardships was successful in reaching England. He was again re-examined before becoming active in the Czechoslovak Squadron attached to the R.A.F.

He took part in the Battle of Britain and in mid October, 1940, while a gunner on a plane over Brussels and while taking part in a power dive, he suffered a sudden onset of pain in the chest, nausea, shortness of breath, and a shock reaction as indicated by cold perspiration and a sensation of extreme weakness. He apparently did not lose consciousness. He was admitted to a Naval hospital in the south of England where a diagnosis of ruptured diaphragm was made. The urgency of his symptoms subsided and he was informed that, although operative interference was required, the surgeons in England, because of air-raid conditions, were unwilling to undertake fancy surgery.

He remained inactive until about mid summer, 1941, when he flew to Canada in a bomber. During the trip he again suffered from acute symptoms of pressure in the thorax and upon his arrival in Montreal was referred to me for treatment.

* From the Montreal General Hospital, McGill University.

He looked ill, and stated that he had lost about twenty pounds in weight in the interval since October, 1940, that he had attacks of faintness and suffered from a loss of appetite with nausea but apparently no vomiting.

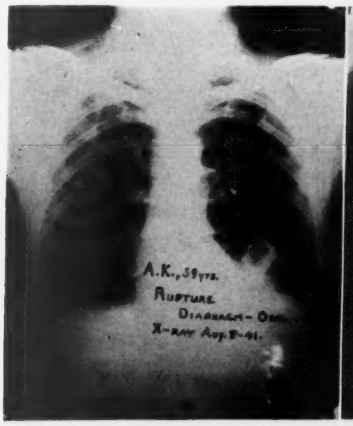

FIG. 1. Flat plate shows hernial sac above the heart shadow.

FIG. 2. Barium filled stomach. The organ lies high in the thorax and demonstrates a typical "upside-down" appearance.

He carried with him a flat plate of the thorax which showed the presence of a hernia occupying the greater part of the left chest reaching well above the arch of the aorta. Examination of the chest showed parts to be tympanitic, other parts to be dull; there was an absence of breath sounds over the left side of the chest except for a smallish area behind and above. (Fig. 1.)

He was admitted to The Montreal General Hospital where further *x*-ray examination, following barium administration, proved the stomach to be located well above the arch of the aorta and proved, also, that the splenic flexure was close to the top of the chest. (Figs. 2 and 3.)

Operation was recommended and eagerly accepted. It was carried out August 17th. Thoracotomy was accomplished through the bed of the seventh rib which was removed from the transverse process to its anterior end. The sixth and eighth ribs were cut so that adequate exposure within the chest was easily obtained. Under the posterior part of the opening a thin layer of completely atelectatic lung was discovered. Under the anterior two-thirds or more of the incision I came directly into an enormous sac. In the cavity, which gave the impression of being about the size of the operator's head, was situated the whole of the stomach, practically the whole of the transverse colon to which was attached the great omentum, the splenic flexure and much of the descending colon. Numerous coils of small intestine were present as were the spleen and the left lobe of the liver.

All of these structures were easily pushed down into the abdomen through the very large hiatus except the stomach which was adherent to the upper and posterior part of the sac over an area about the size of the

FIG. 3. Barium filled splenic flexure is shown lying close to level of the clavicle.

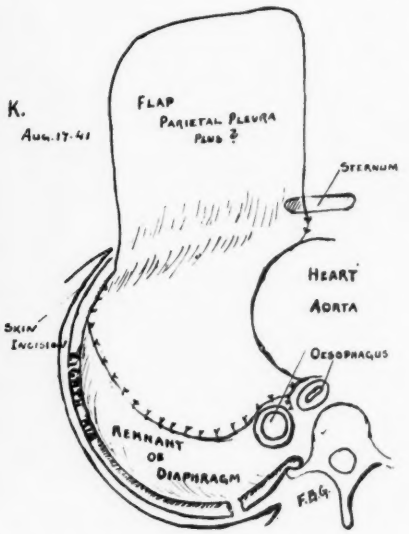

FIG. 4. Diagram drawn at operation illustrates the size of the opening in diaphragm and the method adopted to fill the defect.

operator's hand. I must admit that while engaged in the somewhat trying problem of separating the stomach from the upper part of the sac I felt rather embarrassed since it did not seem possible that I should be able to close the enormous opening between the abdomen and the thorax.

Once the abdominal viscera had been replaced in the abdomen I was left with what I estimated to be about one-quarter of the diaphragm in position in the posterolateral corner and with an opening roughly circular in shape about 20 to 25 cm. in diameter.

If it were not possible to close this opening, the patient's life would have been jeoparized without profit. I decided, therefore, to separate the parietal pleura from the endofascial layer over the anterior aspect of the chest. Incisions were carried vertically up through the parietal pleura to close to the dome of the thorax and joined by a transverse incision. The flap thus outlined was comparatively easily separated from the underlying fascia and periosteum and was found to have considerably more stability than I had expected. It was evidently composed, I believe, not only of parietal pleura but, in part at least, of peritoneum and in all probability of shreds of diaphragm. This adequate flap was turned down and sutured into position to the remnants of the diaphragm and somewhat carefully about

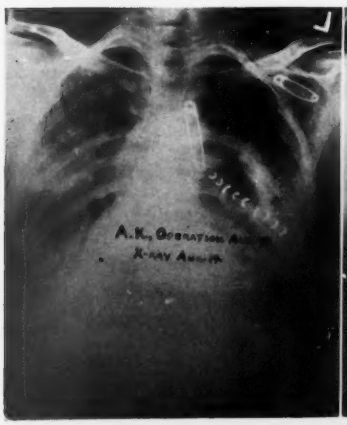

FIG. 5. *X*-ray film forty-eight hours after operation. Absence of any pathological lesion on the left side and atelectasis of right middle lobe is clearly seen.

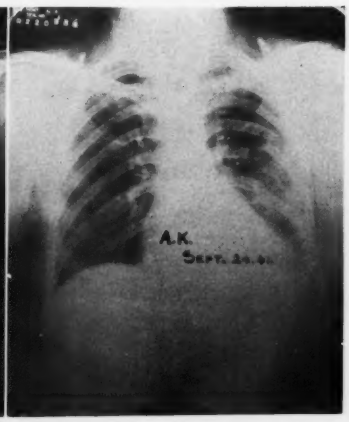

FIG. 6. *X*-ray film five weeks postoperatively.

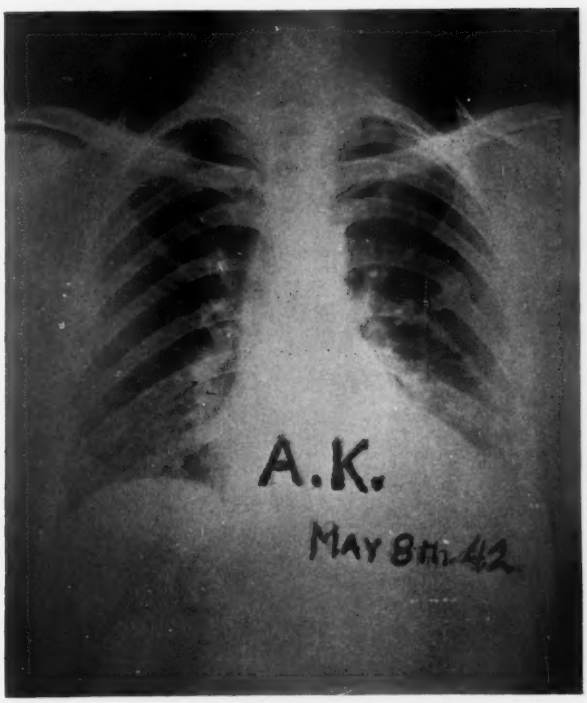

FIG. 7. *X*-ray film eight months postoperatively.

the esophagus and aorta. For this purpose interrupted black silk sutures were employed.

At the completion of the operation the inside of the chest looked shipshape although the lung showed no tendency to come down and fill the

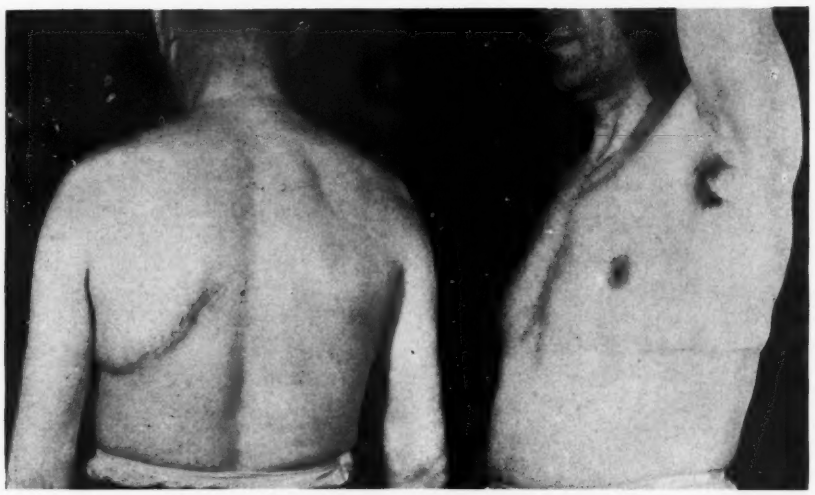

FIG. 8. Photographs of patient eight months after operation.

cavity. (Fig. 4.) Although absolutely no movement of the slip of diaphragm present was exhibited, the phrenic nerve was crushed as it lay on the pericardium.

The wound of the chest wall was closed with interrupted fine black silk sutures with silkworm gut sutures and clips to the skin. Four heavy catgut sutures were employed to approximate the sixth and eighth ribs.

Following operation the patient's condition remained very satisfactory although he developed a localized area of atelectasis, probably in the middle lobe, on the contralateral side. (Fig. 5.) He was kept in an oxygen tent for two days and thereafter rapidly improved in his general condition. Aspiration of the left side of the chest on the second day resulted in the recovery of 400 cc. of slightly blood-stained fluid. He was out of bed on his tenth day and discharged from the hospital on September 11th. (Fig. 6.)

He was examined May 8, 1942. At this time he had put on forty-nine pounds weight since operation. He exhibits no digestive disturbance whatever and does not suffer from shortness of breath upon exertion. He returned to duty with the Czechoslovak Air Force Training Center in Canada on October 15, 1941. His only complaint is that he has been "grounded." Although I have advised against battle aviation, I am certain that he will demand that he be permitted to fly with such insistence that his superior officers will give in. Examination of the chest showed absolutely no lag on the operated side and normal breath sounds are heard throughout. *X*-ray examination of the chest showed no abnormality. (Fig. 7.) There is no disfigurement of the torso and no disability. (Fig. 8.)

SUMMARY

A case is reported of a middle-aged aviator who during a power dive ruptured the left diaphragm. Complete destruction of at least three-fourths of the organ occurred. Massive herniation of abdominal contents into the thorax took place.

Reconstruction of the missing diaphragm was accomplished by a method which I believe to be original. For this purpose an enormous pedicled flap consisting of parietal pleura was employed.

DISCUSSION

WILLIAM J. BRICKLEY (Boston, Mass.): I am privileged to be invited to discuss this case. This was a difficult job well done, and with a good result.

I am a medical examiner. I have been for twenty-two years. My patients give me no history, and mostly my work is by deduction. They cannot tell me, so that I have to deduce a great deal. However, I have been a surgeon. I have examined 90,000 of the living and I have only taken care of 10,000 of the dead so far. I do autopsies and I deal with the big hospitals. I love surgery, I love medicine, and I talk with the fellows that do it. Dr. Gurd's was a good job. That is the first thing I would say.

Next, I am going to discuss the surgery of it. I am going to tell you a little bit about hernia, its diaphragmatic formation. I have talked many times to my own group about it. Hernia is produced, as I see it, by a weakness and then a force. Almost never do I see a hernia produced by a force in an uninjured or a congenitally strong belly wall. I recall the case of a man who jumped off our Customs House, came down 400 feet, landed on concrete; all his bones were broken. He was broken the way you throw an orange against the wall. He had no hernia; his liver was ruptured; all his bones were fractured.

I remember an aviator who came down 500 feet and hit the ground. They do have diaphragm ruptures, and have them very frequently, and I see them I would say two or three in every hundred autopsies that I do. They are not unusual.

The place that the diaphragm ruptures is the left side, either anterior or posterior, and there is a mechanical reason for it. I see the diaphragm so often it is before me as I think of it. At eleven o'clock I was poking my finger through the esophageal opening of a man who had died, so that I would know what I was talking about.

The diaphragm is higher on the left side than on the right side. The left side comes up to the sixth rib, and the other side, the right, comes down to the fourth. The diaphragm on the right side is a little heavier than the diaphragm on the left side. The diaphragm on the left side has a larger plaque. It is a central tandem. You remember there is a right and a left and a central, that is the clover-leaf type. The crust on the left side is not

as good as the crust on the right side. The crust on the left comes down to the sixth lumbar. The crust on the right comes down to the first, second and third lumbar.

There are three holes in the diaphragm. I am speaking mechanically and perhaps I can speak figuratively. The one on the right is the vena cava. The vena cava of the ordinary person is about the size of a broomstick. It would be about an inch or an inch and a quarter in diameter. When there is internal pressure, it is exerted by the liver. The liver is soft and squashes up against the diaphragm and tends to shut off the inferior vena cava. You know when they operate upon the gallbladder, they pull it up, and I wondered why they did. I know. It is because the upper part of the inferior vena cava goes through a deep groove.

On the left side we have the esophageal opening which is near the spine. The level that is shown in a diagram is a higher level than is shown in the diaphragm. Quite often that is so, because the esophagus comes down the center as far as the fourth, and then it begins to go off toward the left, and along about the eighth to the tenth, it begins to leave and go forward and to the left go through a hole in the diaphragm.

The lower end of the esophagus has a sphincter and it is a sort of cuff-like sphincter. There are three of them, top, middle and lower, but the lower not only shuts it this way but it puckers it up against the hole in the diaphragm so that it forms a plug, just the way we would repair a hernia and we would pull something in to plug it. Within the chest you have a negative pressure, within the abdomen you have a positive pressure, so if the hole through the diaphragm at the esophageal opening is large enough, you will tend to come out, first by suction from above and next by pressure from below.

The esophageal opening would admit my finger, but it has to be big enough to have a bolus go through it. Of course, it will always vary, but it goes through at a double angle, through a curved surface and from front to back, going outward and downward. Now it goes through the muscular part in the back of the diaphragm, near the central raphe, but that central raphe on the left is fortified by the pericardium, and hence it is hard to tear that. It is a good central raphe so that you will not get a tear at or around the pericardium unless a person is shot. You will not get it by stresses or strains because it is heavy, but you will get a tear of the muscles on the left side, the muscular part of the diaphragm along the left border, either where it is attached or at its curvature.

The liver is one of the most variable organs we have in shape, size and contour. Sometimes the right lobe of the liver would point up steeply. It would be like the top of a derby hat, and the left lobe of the liver would be like a brim; it would be an inch or two, only, and you may or may not have a left lateral ligament. You have five: right, left, central, coronary and falciform to hold it in place. The right ligament is always good, and the coronary is always good. The left one, however, may be absent. The left

lobe of the liver would be as thin, sometimes, as my finger, and the other part of the liver would be a dome that would run up high, the whole liver being completely within the right chest.

Further, in order that the esophagus may pass down through the diaphragm, the left lobe has to be anterior. On the right it will not come down. That forms a weak place so that with any intra-abdominal pressure the epicardium on the right is safeguarded; on the left, no. It is out of the way. It is either small or is in advance of the hole.

The esophagus itself has a cuff-like lower sphincter and that varies very, very greatly, and the stomach is not as the pictures are drawn. That is, you have not a big cardia with a large end in it. That only occurs after death or when you have a lot of gas. In many stomachs that we see, the esophagus comes down and there is only a little cone until it is distended. I can always drag the end of the stomach or the part of the stomach adjacent to the esophagus into the chest cavity. You do it easily. That hole is like a button-hole, and it is so that when the diphragm pulls, it is shut off. You cannot swallow and breathe at the same time. That is the reason, because your diaphragm shuts it and it will not go through when you pull it down wrong, but if you pull it by inadvertence or accident or otherwise, the esophagus gets above the diaphragm—that is if there is slack enough to do it—and you are in trouble for the esophagus itself is thicker, much thicker than the adjacent part of the stomach, though the esophagus spreads out in the wall of the stomach in a trumpet shape and it is thickened there. If you pull it past that thickening, it stays there, so that often, in doing an autopsy, we find the cone of the stomach has come up into the thoracic cavity, maybe an inch or two or three inches.

Now given that condition, intra-abdominal pressure will tend to make it worse. The peritoneum does not stretch much but it stretches a little. It will not stretch as big as your head. When you are sewing up a fat wall of belly and you are pulling the peritoneum over and you take a stitch, it will tear out if you put too much tension on it. In the hernias we see, they are formed with the peritoneum; they are formed little by little with the gradual stretching, and then, as many of us know, you can have a stretch as big as your head. It will hold an ovarian tumor. We see it in the ventral tumors as big as your head. Therefore, my deduction in the matter is that there is first a weak place, and then a forcing out. It is little by little on top of a small defect.

What are you going to do about it? I would suggest to those who see aviators with that condition, which has been determined either by *x*-ray or otherwise, that they think twice about it. They should compress his abdomen to see whether, under such compression, he might go up; for when he comes down or decelerates, as they call it, that is, comes to the low part of his flight and increases his abdominal pressure, something is likely to project above the diaphragm. You will never project it there because you

have an adequate negative pressure, but you always have a positive pressure pushing it up, so I would suggest that it might be wise to have such a man examined.

I know that I find many of them. About 2 or 3 per cent of those that I see have some sort of cone-shaped projection, and I am only speaking from 2,800 autopsies, so there is something to it.

The manner of it, I will not take up; that is, the force necessary to develop it, but as a mechanical proposition the left side of your diaphragm is not as good as your right side, and in case of pain on the left after an event of this sort, I think it would be wise to think of hernia of the diaphragm.

FRASER B. GURD (closing): I might just say that you will all have recognized that I resisted the temptation to have diagrams drawn with arrows pointing in different directions to show how this rupture took place.

ANESTHESIA FOR THE SURGERY OF TRAUMA

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HAD we discussed anesthesia for the surgery of trauma five years ago, we would have considered the increasing number of traffic, industrial and home accidents. Today, however, we must in addition to these, concern ourselves with the casualties associated with the world wide conflict in which our country is now involved. In this regard, we find our problems quite different from those of the first world war in that civilians as well as our armed forces are subject to attack.

We are confronted with a problem quite different from the last war, namely, that of civilian casualties, besides the wounded soldiers. Developments in anesthesia during the past ten years have contributed much to the improved care of civilian casualties in peace time. First the physician anesthetist has trained himself, not only in the improved anesthesia technic, but in the evaluation and treatment of the patient's general condition and in this way is able to relieve the surgeon of much responsibility. For instance, of great importance is the consideration of shock in these patients who have been subjected to loss of blood, trauma to the nervous system and tissue damage. This is treated with plasma, venoclysis, or transfusion before any anesthesia or surgery is considered. The tissues of the patient, who has recently been in shock, have already suffered impairment of function from anoxia. It is highly important that the anesthetist keep this in mind and furnish the highest possible concentration of oxygen during the operation. Preliminary preparation alleviates the fear of the anesthetic and operation, thus reducing the metabolic processes so that the anesthetic when given is made more efficient and safe. The drugs used for premedication are morphia and atropine for ether, pentobarbital sodium with atropine or scopolamine, for the gas mixtures and spinal. This reduces the incidence of difficult induction, irregular course of anesthesia and has provided better relaxation and postoperative recovery.

Today we have a wide choice of anesthetic agents and their combinations, while not many years ago we were limited to ether for routine use. After much trial and error the choice of drugs and methods has become well established. This has been brought about by the recognition that efficient anesthesia demands a balancing of drugs, anesthetic agents and oxygen. Carbon dioxide absorption, brought forward by Waters in 1926, has revolutionized the administration of gases. In 1929, spinal anesthesia was re-established on a more scientific basis; as a result we may now secure relaxation with safety in the most difficult abdominal procedures and operations upon the lower extremities. In 1930, avertin was introduced as a rectal anesthetic. Since 1934, the rapid acting intravenous barbiturates, evipal sodium and pentothal sodium have been developed as general anesthetics for short operations. Since the re-advent of spinal anesthesia physician anesthetists have utilized combinations of all these various anesthetics with oxygen.

Regional and intratracheal anesthesia have been perfected in the last ten years to a high degree. They find their greatest usefulness in the acute surgical emergencies in the poor risk patients, and in injuries of the head, chest and abdomen.

These are the actual choices of anesthetics for the civilian casualty in peace time: *First, intracranial injuries:* If the patient is in poor condition, regional anesthesia is used supplemented with a 2½ per cent solution of pentothal sodium intravenously with oxygen. In better risk patients, intratracheal ether and oxygen are employed. *Second,* in injuries involving the respiratory tract and oral cavity, including chest injuries and upper extremity surgery, intratracheal cyclopropane is used with carbon dioxide absorption. In poor risk cases, regional anesthesia and oxygen is employed. For abdominal and lower extremity operations, in good risk patients, spinal anesthesia is the anesthetic of choice. In border-line risk patients, balanced spinal anesthesia is preferred. By balanced spinal anesthesia is meant a combination of well chosen premedicating drugs followed by a minimum dose of spinal agents supplemented by a low concentration of cyclopropane with oxygen. This combination preserves, as nearly as possible, the normal physiological reactions of the patient and eliminates the danger of large doses of spinal agents. In the bad risk patient, when spinal anesthesia is contraindicated, small doses of premedicating drugs followed by gas-oxygen with or without regional anesthesia are used. This prevents further depression of the patient's vital functions.

We now come to consideration of the civilian casualties in war. In general, they are treated in the same manner as the civilian casualty in peace time. However, when catastrophes of any magnitude occur, it is necessary to handle large groups of casualties with limited personnel and equipment. This requires preliminary organization well in advance of the anticipated event. Morphia and barbiturates will be given at first aid stations. Cases of gas poisoning with irritation of the lungs will of necessity require regional anesthesia or 2½ per cent solution of pentothal sodium intravenously with the administration of oxygen for respiratory difficulties. No volatile inhalation anesthetic will be used in these cases.

Anesthesia for the wounded soldier presents a number of very complex problems. To meet these problems, too few physicians have been trained in the more improved methods of anesthesia. However, the army and navy are taking vigorous steps to overcome this need. At the Naval hospitals physician anesthetists have been organizing anesthesia along the lines of their experience in civilian practice. For the treatment of naval personnel in the sick bay itself, the risks of explosion and the lack of help narrow the choice of anesthetics to nitrous oxide, pentothal, regional and spinal anesthesia. The Surgeon General's office is now introducing the fundamental policies and organization as outlined by Tyne, Nichols and Wiggin. Specialists in anesthesia have been assigned to the various army hospitals and units to train a personnel for the care of military casualties. Others have been assigned for further specialized training at graduate teaching centers.

These are the procedures to be carried out for pain relief at the various stations of evacuation in the field of operation. At the battalion aid and collection stations morphia and rapid acting barbiturates will be administered for pain relief by medical officers. In the surgical trailor hospitals associated with the surgical hospital, the treatment of shock will be instituted by the anesthetist in charge. Minor emergency operations will be performed with intravenous pentothal sodium and nitrous-oxide-oxygen with a portable carbon dioxide absorption apparatus. Spinal anesthesia may be used at this unit if a move is not anticipated within twelve hours. At the evacuation hospital, where the major part of the surgery will be carried out, cases of shock will receive the attention of the physician anesthetist in charge, and here the patient will be given essentially the same anesthetics and methods of introduction which are employed in the general hospital, including preoperative and post-

operative care. At these hospitals will be stationed the neurosurgical, plastic maxillofacial, and thoracic surgical teams with a physician anesthetist in charge of each one. The physician anesthetist will be selected according to his ability. For example, an anesthetist proficient in intratracheal anesthesia will be assigned to the thoracic surgical outfit. In many cases one officer anesthetist may have to supervise several operations at the same time. In such an event he will depend on especially trained anesthesia assistants.

The general hospital in the zone of the interior is the final unit in the line of evacuation to which the wounded will be finally evacuated. Here the same facilities for anesthesia which exist during peace time will be available.

There is a good deal of controversy regarding the ideal anesthetic. The variations in the human organism are so multiple that it is still a remote possibility. An anesthetic is as safe as the anesthetist. The safest anesthetic is the one with which the anesthetist is the most familiar, and that in which he has been trained. The patient in anesthesia has been the forgotten man and there has been committed too frequently the error of fitting the patient to the anesthetic rather than the anesthetic to the patient. More and more must the well trained anesthetist be a practicing physiologist. Only in this way can he care for the varied needs of the patient.

We have in the wounded soldier a patient who is different from the civilian in that he is robust but on the other hand under a very considerable degree of psychic tension. His condition may be complicated with alcohol, tobacco or a full stomach. He may be in varying degree of shock for which there is need of special care without the facilities of civilian practice. The treatment of shock is the first consideration. Then, in the choice of drugs, morphia will be used for relief of pain on the battlefield where anesthetics are not available. Scopolamine would add to the efficacy of morphia under these circumstances. It was discovered in the last war that the majority of the wounded soldiers could tolerate much larger doses of morphia than the civilian patient in peace time because of their rugged physical condition.

Barbiturates will be used to allay shock, to relieve fear and apprehension, in the field of action and also for premedication and intravenously at the surgical hospitals. Chloroform will be used only under the most adverse circumstances and then only by the expert anesthetist. In the hands of the inexperienced sudden death may occur. The same applies to ethyl chloride. However, in the

hands of the physician anesthetist, familiar with its use, it is a fairly safe agent for the induction of ether, or for short surgical procedures. *Ether* is the safest anesthetic in the hands of the occasional anesthetist. It has the widest margin of safety and applicability. Its disadvantages as stated by Kaye, of the Australian Imperial Forces in the Middle East, are concerned with the difficulty of induction, the need for increased personnel, more prolonged postoperative care, and the incidence of respiratory tract irritation, nausea and vomiting.

Nitrous oxide will be most useful for short operations and as preliminary to the induction of ether. The handicap of heavy cylinders and its manufacture makes it difficult to transport and to obtain. To produce complete relaxation of the abdomen nitrous oxide is not sufficient without adding ether. According to the Bureau of Mines this mixture is more explosive than cyclopropane.

According to Lundy the quick acting barbiturates administered intravenously will be used more often than any other type of anesthetic. Pentothal sodium in 2½ per cent solution should be used only by experienced physician anesthetists. Induction is rapid and the recovery early, making it of great value in this fast moving type of warfare. Rectal avertin will find but little use in this type of surgery except as the civilian general hospital. The use of cyclopropane-oxygen will be limited to the evacuation and base hospitals because of its explosive properties. This gas mixture is unsurpassed as a supplement to spinal anesthesia because of its high oxygen content. For the same reason it is unequaled as an anesthetic in thoracic operations. Regional anesthesia, either local infiltration with novocaine or plexus block, will be used wherever possible to alleviate the necessity of rendering the patient unconscious when this would react badly upon his general condition. Kaye reports that the wounded patient does not prefer it and that it makes operating difficult for the surgeon. However, if it is indicated the psychic factors of the patient can be controlled by intravenous barbiturates and gas and oxygen. There is no comparison between the use of spinal anesthesia in World War I and its present status.

Among the U. S. forces in this war spinal anesthesia will be used more frequently than any other method in operations below the diaphragm. The agents will be limited to novocaine and pontocaine. It will be of great benefit to both the patient and the surgeon in war because of its quick induction, the complete relaxation which it affords, the absence of interference with laryngeal reflexes, the

low incidence of nausea and vomiting and the early recovery with very low morbidity postoperatively. Even the patient in shock can tolerate a low dosage of novocaine and pontocaine for operations below the hips. It may be found feasible in severe injuries to the lower extremities to administer spinal anesthesia to patients so injured for relief of his pain and shock even though operation will not be performed immediately. There is the added advantage of spinal anesthesia in that one medical anesthetist may administer it to several patients and supervise them all with the aid of trained technicians.

In conclusion, there has been presented a plan for anesthesia for the surgery of trauma for the civilian casualty in peace and in war and for the wounded soldier.

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DISCUSSION

GUSTAV F. BERG (Pittsburgh, Pa.): There is one type of anesthesia that I should also like to recommend to you, and that is spinal anesthesia. I have been using spinal anesthesia since 1916 and I think it is one of the greatest anesthetics we have ever had. I will not discuss sodium pentothal because Dr. Wagner knows a great deal more about that than I do.

J. HUBER WAGNER (Pittsburgh, Pa.): I enjoyed Dr. Wiggin's paper very much and think it gives us all a lot of food for thought. One statement is quoted from Lundy, that he thinks the coming anesthetic in wartime injuries will probably be sodium pentothal. I believe that when we become better acquainted with this drug, we will use it more. I might say that in the past four and a half years we have used it rather exclusively in our clinic at Pittsburgh, having over 7,000 cases, and as yet have not had even one postoperative pneumonia.

Lundy says it is good for short anesthetics. I would like to disagree with him there. We have used it in some cases as long as two and a half and three hours, using a maximum amount on one occasion of some 40 gr., with the patient recovering.

I believe that not only physician anesthetists have to be trained in this, but I think our female anesthetists as well can be trained so that they can give it without any danger to the patient, and I would ask your indulgence in the use of pentothal for your patients in the future.

BELFORD S. LESTER (Birmingham, Ala.): May I ask Dr. Wiggin about the radiating pains that follow in certain cases of spinal anesthesia? It does not seem to make very much difference whether those are administered in the army or the naval hospitals. They bother the patient from two to six, eight and ten weeks after the spinal anesthesia has been administered.

Secondly, how about the toxic hepatitis after a cyclopropane anesthesia that comes on anywhere from eight to ten days after?

SIDNEY C. WIGGIN (closing): I hold no brief against ethylene except to say that the theoretical proportions of oxygen with the gases are 90 and 10 for nitrous oxygen, 80 and 20 for ethylene, and 15 per cent cyclopropane and 85 per cent oxygen. In our experience, the cyclopropane, I think, has proved better than ethylene, although ethylene was a great improvement over nitrous oxide.

Dr. Wagner referred to female anesthetists. I suppose he means nurses. I know that the Army is training some nurses in intravenous anesthesia, but I believe that there is an element of danger, because when a patient goes bad with intravenous anesthesia, it really requires the attention of an expert anesthetist.

Regarding the radiating pains that Dr. Lester mentioned, which occur after spinal anesthesia in both legs two, four, six and eight weeks postoperatively, I have not had that complaint among my patients and I have used it since 1928.

As for the toxic hepatitis, I have used cyclopropane for the better part of ten years and I cannot recall any complication of that nature among my patients, and I have not heard it reported.

SUBTROCHANTERIC FRACTURES OF THE FEMUR*

AN OPERATIVE APPROACH FOR OPEN FIXATION

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SUBTROCHANTERIC fracture of the femur is one of the major problems in the management of fractures. It is most commonly due to direct violence and is distinctly an accident of middle age and late life. In the series which I have analyzed there are forty-two cases. Of these there was only one in each of the first, second and third decades, while there were thirty-nine past forty years of age. The distribution of age was as follows:

Age	No. of Cases
1–10	1
10–20	1
20–30	1
30–40	8
40–50	8
50–60	4
60–70	7
70–80	8
80+	4
Total	42

These figures indicate that this fracture is an accident in which the passing of youthful agility is a large factor.

The portion of the femur in which the fracture occurs is encased in a large number of muscles, each of which is attached to the femur at one end and exerts some measure of control over the movement and fixation of this bone. The multiplicity of these muscles and the varied directions of their pull account for the marked displacement of major fragments as well as for the bizarre scattering of smaller pieces of bone. Because of the many small muscles which pass in a more or less transverse direction between the pelvis and the femur, and the proneness of fracture lines to be oblique or fragments to be

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comminuted, it is easy for muscle to become interposed between fragments and thus interfere with reduction and delay union.

The characteristic displacement of fragments and its mechanism

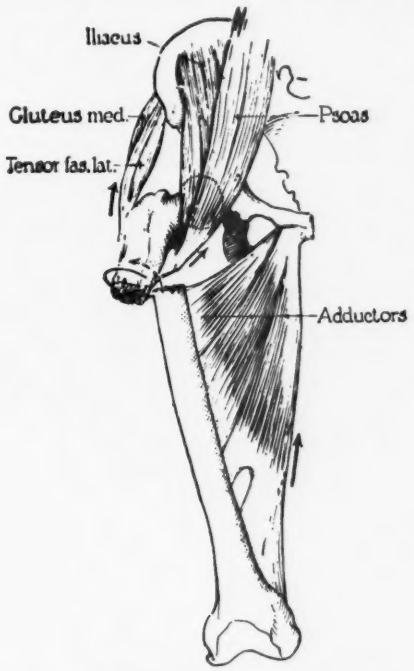

FIG. 1. Diagram showing displacing effect of muscles in fracture at junction of upper and middle thirds. (From Magnuson's Fractures. Philadelphia, J. B. Lippincott Co. Drawn by W. C. Shepard.)

of production is as follows: The proximal fragment lies anterior and lateral to the distal one, having been rotated outward and flexed by the psoas and illiacus muscles and abducted by the tensor fascia lata and gluteus medius muscle. The adductors draw the distal fragment inward. I know of no better illustration of this than the one from Paul Magnuson's book on fractures. The upper fragment is so short that it is not possible to influence its direction by leverage or to rotate and fix it. Consequently, it is necessary to place the distal fragment in line with the proximal one. Where the line of fracture is transverse the fragments may occasionally be engaged by manipulation under general anesthesia, but when the line of fracture is oblique or the fragments are comminuted, reduction is rarely possible and position of fragments must be maintained by traction. Some form of balanced traction is generally advocated in which the leg is

rotated externally, the thigh is flexed and abducted, and the knee is flexed to relax the hamstring muscles. Russell's traction or suspension under a Balkan frame with traction by means of a Thomas splint with the Pearson attachment are methods most commonly used.

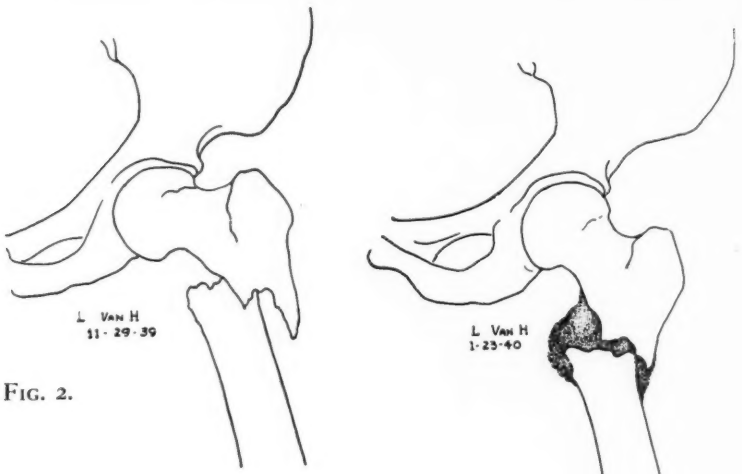

FIG. 2.

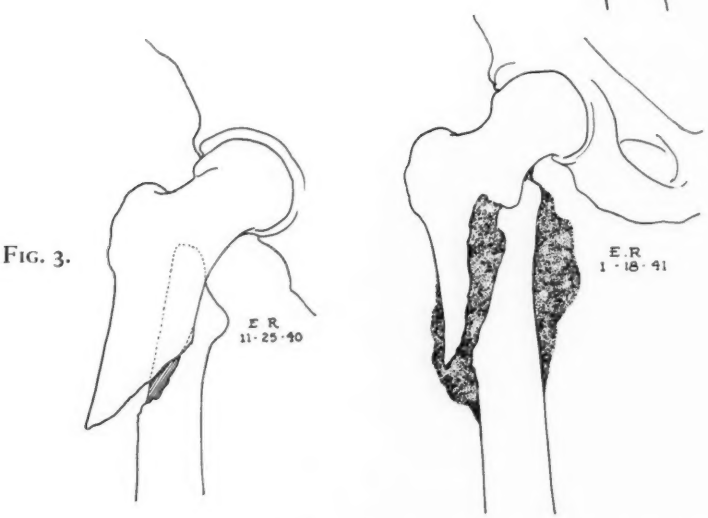

FIG. 3.

FIG. 2. Usual displacement and result after callus had formed. (Tracing from *x*-ray.)

FIG. 3. Position during traction and final union. (Tracing from *x*-ray.)

When traction fails to effect a satisfactory reduction there will be a high incidence of delay in union, angulation and shortening. In such cases we believe that many of these breaks should be subjected to open fixation at an early date. Formerly, we were inclined to temporize because of the magnitude of the operation, but with the approach, which I shall describe, our attitude toward the problem

has been considerably altered, even though the fracture is frequently associated with such unfavorable factors as senility and obesity.

The common approaches are (1) the anterior one described by

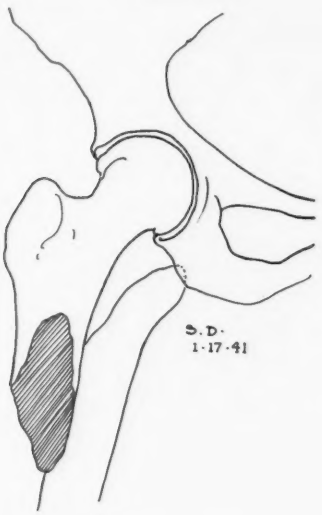

FIG. 4. Position during traction. (Tracing from *x*-ray.)

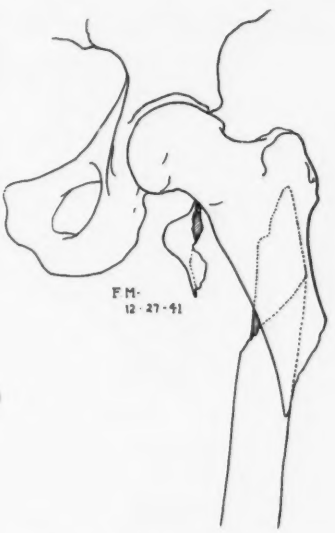

FIG. 5. Position during traction. (Tracing from *x*-ray.)

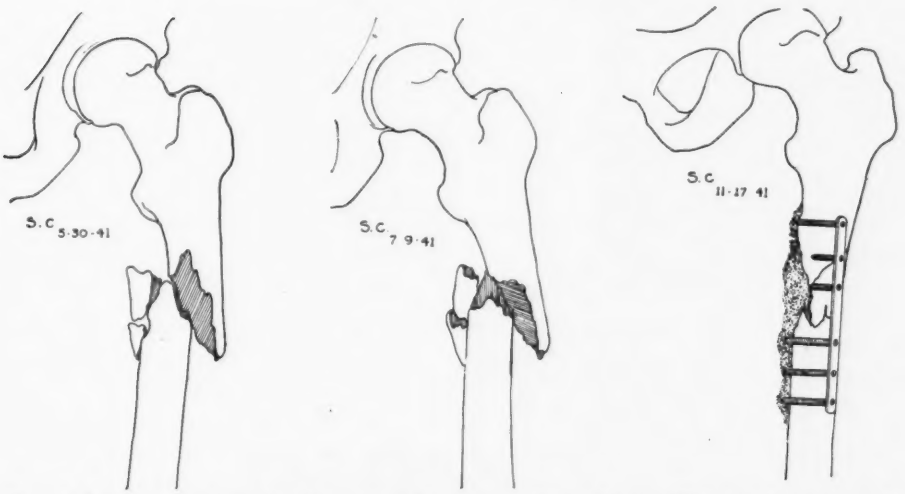

FIG. 6. Position and result when operation was performed late. (Tracing from *x*-ray.)

Henry in which the incision follows a line from the anterior superior spine of the ilium to the outer border of the patella, and (2) the lateral one in which the incision extends downward from the great trochanter. In both of these the bone lies very deep and must be discovered through the fibers of the vastus lateralis muscle. I know of few more harrassing operative procedures than trying to expose

and mobilize fragments of the upper end of the femur in a muscular patient through either a lateral or anterior approach.

The approach which we have employed in six cases has made the

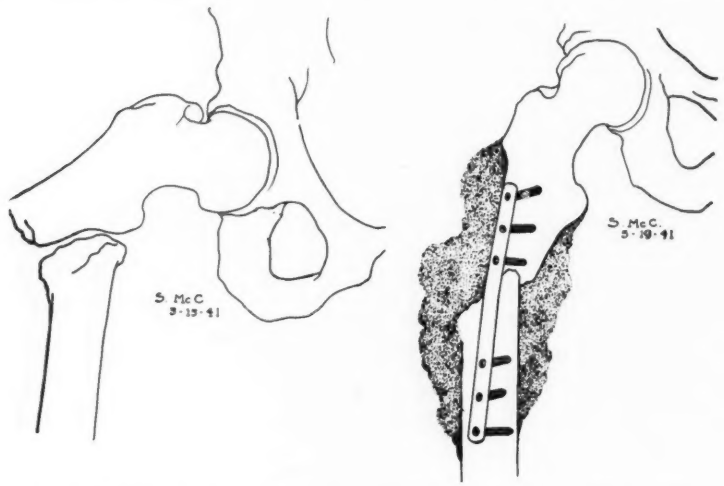

FIG. 7. Position and result when operation was performed late. (Tracing from *x*-ray.)

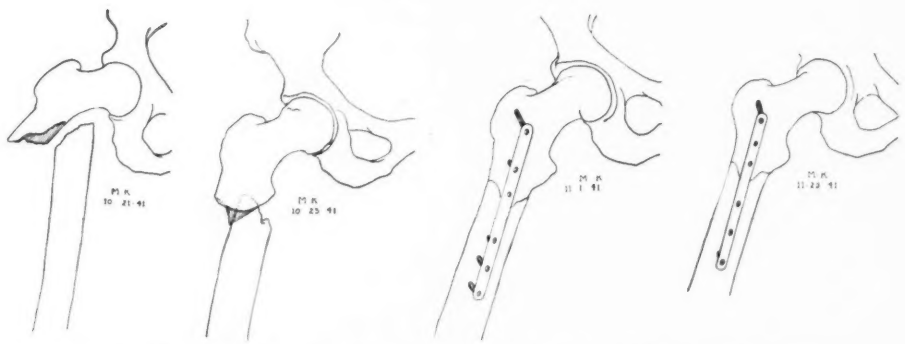

FIG. 8. Result when operation was performed early. (Tracing from *x*-ray.)

operation much easier for the reason that the muscles can be separated, the bone is nearer the surface and structures which must be avoided, particularly the sciatic nerve and femoral vessels, are not so placed as to cause one to fear their injury. This operative approach is not original with me, but I do not know to whom to give the credit.

When possible we like to apply a single hip spica to the patient several days before operation. When doing this we place over the operative field several gauze pads about the size and extent of those to be used as wound dressings after the operation. The case is bivalved before it is quite firm and is laid aside to harden and be ready for application at the completion of the operation. By this plan

the anesthesia is not prolonged by the period necessary to apply the cast, and the operation can be performed on an ordinary operating table.

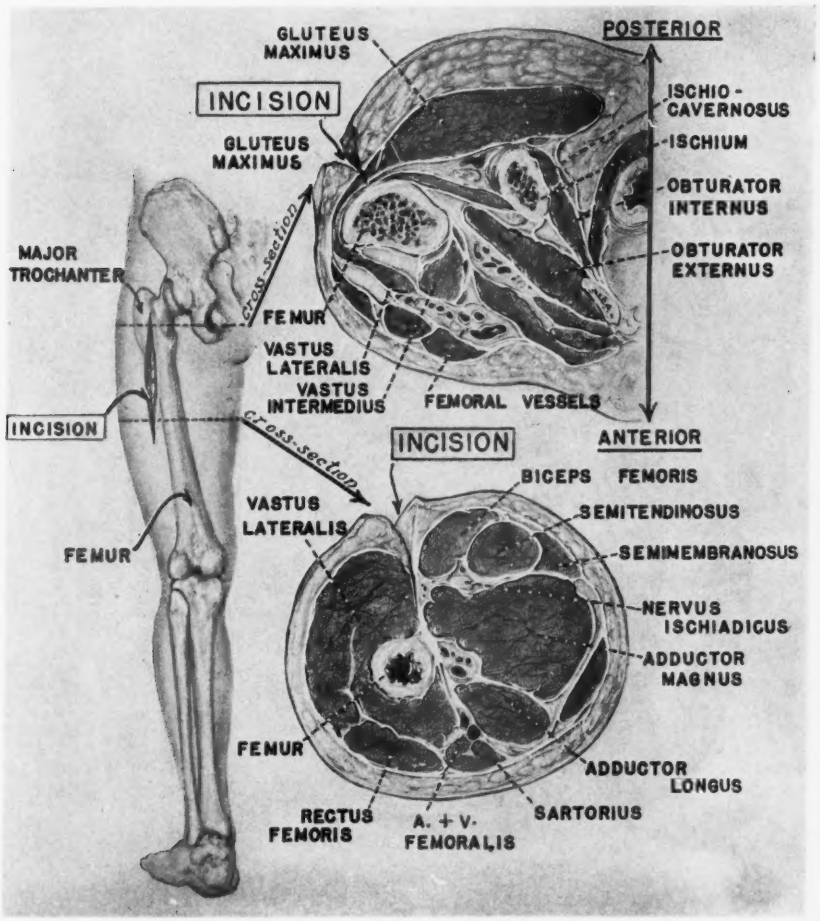

FIG. 9. Cross sections of thigh at levels of trochanter and upper third showing approach to bone by separation of vastus lateralis and biceps femoris. (Adapted from Manual of Surgical Anatomy. Medical Department of U. S. Army and Navy.)

The patient is placed face down on the table and the side to be operated upon is elevated about two inches. The incision follows the posterior border of the fascia lata and begins at the greater trochanter and extends downward eight to ten inches. In its upper portion the insertion of the gluteus maximus muscle into the fascia lata is cut, leaving enough of the insertion to repair after completion of the operation. In the distal portion of the incision after the insertion of the gluteus maximus muscle is divided, the biceps femoris,

which lies on the medial side, can easily be separated from the vastus lateralis by blunt dissection. The femur is then felt at the bottom of the incision. From above downward the attachment of the

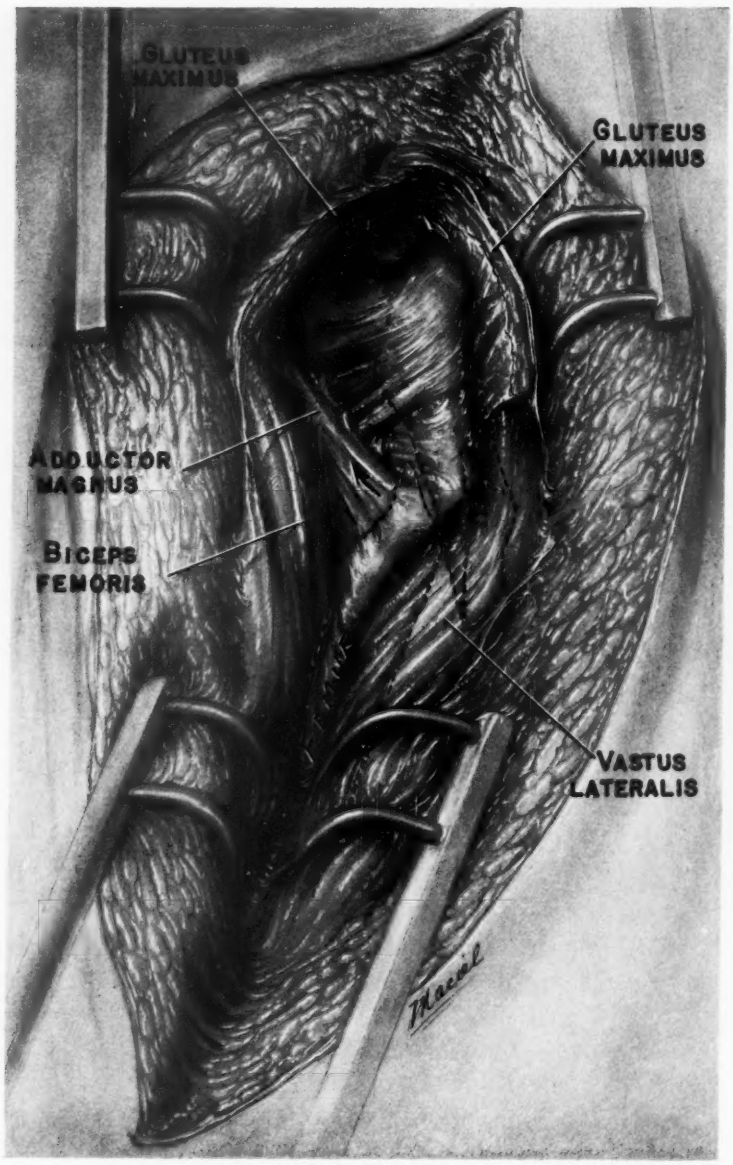

FIG. 10. Drawing showing operative approach.

quadratus femoris and then that of the adductor magnus muscles may have to be separated from the posterior surface of the femur. The proximal fragment usually lies anterior and external to the distal one. Each must be separated from any attachments it may

have formed, then by levering or clamping with bone holding forceps and manipulating, the two fragments are maneuvered into suitable end-on apposition. The method of permanent fixation is a matter of choice. In all of our cases we have used six-hole plates.

Silk is used for ligatures and sutures throughout the operation. After completion of the operation and dressing, the posterior shell of the cast is laid on the patient and he is turned and lifted in this case to stretcher or bed. The anterior shell is then applied and the two are strapped together accurately with adhesive straps.

In all of the six patients operated upon union has been prompt and firm and good function has been restored.

Two of our cases did not come to us until efforts by others had been ineffectual and considerable time had been lost and in two of our own cases we continued traction methods beyond a reasonable period. In the other two cases operation was performed within ten days, and in both of these good useful function was restored in four months. The periods of hospitalization were fifty-nine days and sixty-four days, during which time a cast was in place eight weeks and seven weeks.

Of the thirty-six patients treated without operation both Russell's traction and Thomas splint with Pearson attachment were used; in several cases both were tried. In all cases the traction was followed by the application of a cast. Two patients died during the treatment. In the thirty-four survivors union resulted in all, but two were unable to bear weight before eight months. The maximum shortening was one and one-half inches in two cases, one inch in two, one-half inch in four, and none in twenty-six. All have united by excessive callus.

SUMMARY

The posterior approach offers a much easier and simplified method of operating upon subtrochanteric fractures of the femur. In suitable cases, when a satisfactory reduction cannot be obtained by other methods, we believe that an early use of this procedure will be followed by better results, less inconvenience to the patients and a shorter convalescence.

DISCUSSION

HENRY C. MARBLE (Boston, Mass.): I am very glad that Dr. John Caldwell brought up the subject and I think if we had more confessions of defeat, that it might be the ground for discussion. I do not recall that I

have heard that discussed very much, particularly in wartime. My recollection of that particular injury which Dr. Caldwell brings to our attention as a defeat, is of a through-and-through wound that passes through the soldier's thigh; he sustains a subtrochanteric fracture with comminution and you are confronted with two or three problems. The first problem is, what are you going to do to hold the proximal fragment? The first picture Dr. Caldwell showed by Dr. Magnuson is very simple, but when you have overcome the degree of upward and outward displacement of the proximal fragment and just at the time that you have got the distal fragment to meet it, the proximal fragment goes a little further, and when you get that it goes up still little further. When you end up, you have the patient literally in a knee-chest position in an effort to hold it.

I have never seen one of those cases handled by plaster of Paris fixation, such as is now used, and I wonder how it would manage that proximal fragment. That is always a problem and I recall having some of these men treated with their knees in flexion, their thighs in flexion or better, and dressing the wound on the front of the thigh, and then lying down on the bed and dressing an equally large and sometimes more infected wound in the buttocks. It is a difficult fracture to handle.

There is one other factor in the compound injuries which I think we should bear in mind that this is a source of secondary hemorrhage. There is a large artery which accompanies the sciatic nerve, which is difficult to control, difficult to expose, and having exposed it, difficult to tie. It is also intimate to the sciatic nerve and I have often wondered how many of these cases have bled and the sciatic nerve has been tied with the artery in an effort to control the hemorrhage.

I think this is a very important matter for discussion and I am very glad Dr. Caldwell brought it up.

FRANK P. STRICKLER (Louisville, Ky.): I think Dr. Caldwell has given us a very timely paper and has shown us some excellent results. When Dr. Morrison asked me to discuss this paper, I ran through a series of 100 cases that we had had, and I found essentially the same facts that the doctor has found, namely, that this is an injury of middle life and of late life; but I also found, much to my surprise, that in these injuries under thirty years of age, they were complicated by compound fractures of the tibia and fibula, and these cases occurred in automobile accidents. Of course, a compound fracture of the tibia and fibula complicates this injury considerably.

We have operated upon only 10 per cent of our patients. We use the lateral incision from the greater trochanter down parallel with the femur, and we have done our cases on a hollow table. We seem to think that we can pull it in line better that way and hold it in position that way.

The only trouble that we have had with our cases is that we have found that a large number of them have been rather severely comminuted, and that would make a plating operation rather difficult due to the fact that we simply had nothing for the screws to hold to.

We have used largely conservative treatment, and with very good results. We have used the Thomas splint with the Pearson attachment, and a Kirschner wire through the crest of the tibia just below the tibial tubercle. We put that through under local anesthesia, as a rule, in bed, without moving the patient to the operating room at all. We then apply a Thomas splint that has a rather snug fitting ring. We believe that the ring helps hold the upper fragment and helps to maintain position. We put the extremity up in slight flexion at the knee. This relaxes the hamstring muscle and the flexor muscles. We then apply a weight. I think if you are careful in applying this weight and if you watch your cases carefully with *x*-rays and measurements, you will have no difficulty.

It is very easy to measure these cases, and we *x*-ray our patients not less than every two weeks, and if we have any difficulty with them, we *x*-ray them more frequently than that. I think one of the reasons many of us get into difficulty with fractures is that we are too Scotch with *x*-rays and we do not take enough of them. That is really the only way in which you can tell what a fracture has done. Of course, you do not want to go to the other extreme and *x*-ray them so much that there is a possibility of interfering with bone growth. That is the other extreme, but I believe, from my experience at least, that it is safe to *x*-ray a patient at least every two or three weeks, and that by watching these cases carefully, you can get good results.

I do not know why, but I have not had a great deal of trouble with this fracture and I have gotten very good results. We have had no cases of nonunion and we have had very good functional results.

As far as the incision goes, I can find no fault with the doctor's incision at all. The only objection that I see, or criticism that anyone could offer to this type of incision, is the position and the possibility of its becoming contaminated when the patient eliminates. That is the only criticism that I could offer to it, because we all do have accidents and get incisions contaminated in that region.

I recently had a case that was very interesting to me from this standpoint. Of course, it might not interest you much, but my father saw this man with a fracture of the type that we are talking about, forty years ago. The man fell about a week ago and refractured that femur through the same site, and he came to me forty years later with a fracture through exactly the same fracture that he had years previously.

Robert P. Dobbie (Buffalo, N. Y.): My experience with this type of fracture has been very limited and I really have no right to discuss Dr. Caldwell's paper. All I have to say is that my experience has veen very limited in handling this fracture and I have had my difficulties. Unlike Dr. Strickler, I found it a very difficult fracture to handle in the few that I have handled, and as usual, Dr. Caldwell brings a point that is practical and concise and very valuable. The posterior approach is very intriguing and I am sure I will keep that in mind with the next fracture of this type

that I have to treat, due to the difficulty I have had in my few cases in getting through these large muscles in these very obese patients.

FRED H. SMITH (New York, N. Y.): In many of the subtrochanteric fractures that we have had, we have attempted to fix them by plates, and in some of the very high ones even use a Smith-Petersen nail with a Thornton plate. The purpose of internal fixation is, in the first place, to get anatomical position with rigid fixation which permits moving the patient and allows him to get up. If we get firm, rigid fixation, we believe it is not necessary to use any plaster of Paris afterward, and the patients get a much quicker and a better return of function.

After using a plate—we usually use a six-screw plate—we use one or two hand-fixing screws which are put in at a different angle from the screws holding the plate, and in that means it is not possible to bend or twist the plate at the fracture line. We believe we can start hip motion right from the start, and we also believe the patients can be allowed up in a chair in a short time, and that cuts down very greatly on the possibility of postoperative pneumonia and other conditions that these older people get.

H. GURTH PRETTY (Montreal, Canada): This subject of subtrochanteric fractures, I think, is a very interesting one. It has given a good deal of trouble in Montreal. We run into a large group of them, and our experience has been that by the closed method, we were not able to get a reduction; we got a certain degree of shortening and deformity and we had a long convalescence. Furthermore, lately we have been running into a fair group of compound fractures, and we have found in these compound fractures that our difficulties are even more severe than before, so that on some of them we have been attempting almost immediate plating. In other words, the wound had been débrided, it has been watched closely for a period of a week, with sulfonamides, and using a. t. s. and also anti-gas gangrene serum at the same time; then we have gone in and reopened the wound and replated it. The point I want to take up in connection with this slide is that this was a man of about fifty who was struck by a steel rail. He had a hole in the side of his thigh big enough to incorporate two fists, and no matter how we tried to manipulate him, this somewhat bizarre "butterfly" got in the way and our pieces flew all over the place.

The second thing was the question of holding this bone in position for plating by using some lash screws and six-screw plates, and I found by using a small plate anteriorly with the six-screw plate on the side, we seemed to get a very much better position than by a single plate and lash screws.

GUSTAV F. BERG (Pittsburgh, Pa.): I think that Dr. Caldwell is to be congratulated for bringing before us one of the most difficult fractures that we have to contend with, and I believe that Dr. Caldwell has stressed a point which is very valuable. This is one of the types of fracture that, in my opinion, is always an open reduction case.

There is one thing that you must remember. When you are getting up into the trochanteric region, the bone is soft and you may have difficulty

with your screws having sufficient purchase. In these comminuted fractures it is often a good idea to go back to Dr. John Murphy's theory, doing an intermedullary graft, taking a piece of bone, cleaning out the trochanterous structure about, putting in the bone and putting on your bone plate.

HENRY C. MARBLE (Boston, Mass.): I think there is one matter that we have overlooked. I do not know that it was overlooked, but we always believe, also, that fracture in this particular location is very apt to be metastatic and it is a pathological factor. This seems to be a point of election for certain metastatic cancers, Paget's disease, and we must not forget that in our discussions.

JOHN A. CALDWELL (closing): I am very grateful to Dr. Smith for the suggestion for the management of these cases. Further than that, I think there has been very little discussion of the procedure. Most of the men have given very good reasons for it, which is lack of experience. The purpose of this was to try to popularize experience with it.

It might carry a little further conviction if I would give here a report of one of these operations. This was done by the resident surgeon on the service, a man of extraordinary capability who does everything well, and who writes this report. This was the ordinary, routine, operative report which I find it very difficult to improve on in describing the operation:

"The entire leg was then encased in a double layer of sterile stockinette which was extended as far as possible upward on the posterior aspect of the thigh. The draping was supplemented with sterile towels and with plain sheets in such a manner that the leg was within the field of operation and available for manipulation." That is, the leg, covered with the sterile stockinette, was outside all sheet draping so that it was possible to take up the entire leg without contaminating the field at all.

"A longitudinal incision was made after opening the stockinette in the palpable groove between the vastus lateralis muscle and the biceps femoris. The patient was fairly obese, and about 1½ inches of adipose tissue was cut through before exposing the deep fascia. The approach came directly down on the tendonous insertion of the gluteus maximus muscle, and the lower portion of this was divided close to its insertion. Then by separation between the belly of the vastus lateralis and the biceps femoris, the shaft of the femur was readily exposed without exposure or injury to any major nerves or vessels. The muscle fibers attached to the bone surface were stripped free from below upward to expose the fracture site and the hematoma surrounding it.

"The proximal fragment was displaced anteriorly and laterally and the distal fragment posteriorly and medially with approximately 1½ inches of overriding. The ends of the two fragments were dissected free and grasped with Lambot bone-holding forceps, and by manipulation of these two instruments and the manipulation of the lower leg and foot by an assistant, and with the use of a smooth bone skid, the displaced bones were reapproximated in 100 per cent anatomical reduction. A five-inch Shermann plate of

stainless steel with six holes was now applied to the posterior surface of the bone, and one screw was inserted above and one below after preliminary drilling of the bone. With these screws in place, a Lowman clamp was applied around the fracture site and over the Shermann plate to maintain the two bone ends in accurate apposition while the remaining drill holes were made and the plate fixed firmly in position. Three screws were used above and three below."

That gives in very complete detail, I think, everything that is done with this, and that is about the procedure as it is carried out. I think that comparing this approach and this manipulation with the ordinary lateral and anterior approach is like comparing, as far as speed and effectiveness are concerned, the difference in travel by train and airplane. It is much more direct and much easier.

TREATMENT OF INJURIES OF THE HAND REQUIRING SKIN GRAFTING

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IT is not the author's intention to enter into the fine art of skin grafting as applied to the extensive practice of the plastic surgeons, but merely to call to the attention of surgeons engaged in the treatment of injuries, the importance of *immediate* skin graft in injuries of the hand.

Commonplace injuries of the hand are the stepchildren of surgery, in that they are by far the most frequent of injuries and are cared for by thousands of physicians and surgeons. We accept these injuries and resultant deformities usually as trivial, often because of their being commonplace, which leads to indifferent care. This paper then is a plea for a sharper focus on these injuries and a mobilization of our present surgical knowledge from all sources, toward more intelligent care.

The challenge thrown out by Walter Estel Lee's oration on Fractures and Other Traumas at the Clinical Congress of the American College of Surgeons, Boston, November, 1941, should be a whip and if so accepted may overcome the stigma of the quotation of Dr. Meeker, that, "A general surgeon is one who does not do anything particularly well." The sting should prompt the general surgeon to borrow from the vast field of knowledge of his specialist brothers, and to apply approved methods in the care of injuries of the hand. In so doing he must become a student of pathology, orthopedics, plastic surgery and neurosurgery. The proper marshalling of this knowledge will lead to real intelligence in the care of hand injuries, and the general surgeon may make for himself at least a small spot in the sun and find that he can do some *one* thing particularly well.

The hand as a member of the body has never been more important in the history of the world than at this particular time; this being a mechanical age a resultant mechanical war has ensued. The importance of the full function of the hand is obvious. We forget that we have ten digits on our two hands; we do not have an adequate conception of the value of these members or of their co-related values. A hand without digits is practically a total loss, the hand

might as well be absent. Functionally, the digits may be divided into two major correlating units, the thumb and the fingers, each probably of equal importance. A hand without the thumb is a 50 per cent loss of hand function, conversely, the loss of four fingers is a 50 per cent loss. The relatively greater value of the thumb over the individual fingers lies in its being a member in apposition to the four fingers. The loss of either of the cardinal components of the hand nullifies the palm almost entirely.

The original work of infections of the hand by Kanavel and the continuation of this missionary work by Koch and Mason and others, have entirely revolutionized care of hand injuries and, in fact, have made an expose of fanciful claims of the value of skin antiseptics. At last the fundamental principle of skin cleanliness through the media of soap and water properly applied, has driven from the field myriads of caustic cell destroyers masquerading under the term of antiseptics, many of which were not even antiseptics, and almost all were cell destroyers.

All hand injuries should have no further first aid than a sterile dressing and a tourniquet applied to relieve obvious serious hemorrhage. Pressure control of hemorrhage at the site of the injury generally should be condemned. The injured hand, when seen by a surgeon, should be carefully inspected and tested by direction to the patient as to nerve, tendon and bone injuries. *X*-rays should be taken in practically all but the most simple of lacerations. Fractures are of secondary importance, in that lacerations and soft tissue damage demand first consideration.

The hand should be thoroughly cleansed with soap and the use of gallons of water. Injuries on the palmar surface require a brush; others should be cleansed gently with sterile gauze. In injuries of the fingers, nerve block anesthesia should be employed before careful cleansing of the wound area is instituted. Larger, more complicated injuries of the hand require general anesthesia of a preferred type. No injuries are *ever* repaired with local procaine infiltration. Ideally, all hand injuries should be accorded operating room asepsis. Practically this may not be obtainable, nevertheless, this goal should be set up for future attainments. No surgeon would permit general abdominal exposure without proper masking and aseptic procedure, yet many a hand injury presents far more inviting fields for expired air contamination.

A sterile rubber glove is fitted on the hand, an incision made over the desired area (rubber shortage may preclude the use of this

extravagance) and sterile rolled stockinette used. Hemostasis is essential to correct repair. The skin edges are removed in an intact circle which should include the walls to the bottom of the laceration. Débridement should be thorough but not reckless. The use of powdered sulfanilamide sprinkled lightly over the denuded area seems to be definitely beneficial; however, the dumping of large amounts is certainly detrimental to clean healing. Any type of fine nonabsorbant suture may be employed, and a stitch used which does two things: first, co-apts the skin edges in gentle approximation, and second, does not exert cutting pressure.

By far the greatest number of injuries to the hand are to the fingers and thumb and need not be enumerated. The injuries which require immediate skin grafting are those with extensive loss of skin, usually through crushing injuries and burns. The simplest of these are the slicing lacerations of the finger tips, commonly occurring in contact with jointers, meat cutters and other mechanical cutting devices. When the tip has been excised with the greater portion of the ball of the finger and the distal phalanx exposed, the area should be grafted. These injuries, if not grafted with skin, leave the peculiar toothpick finger which is permanently damaged, particularly for fine work. Close observation of the function of these deformed fingers will show how little they are used. The author has long since ceased to scoff at the patient who brings in the severed portion of the finger. Usually if seen within a short time careful cleansing and application of the severed segment will result in a take.

The wringer-type of hand injury in which the hand has been literally peeled of the skin, requires meticulous care; not infrequently the skin is not lost at all, simply rolled up on the hand. All of these are hospital cases unless operating room facilities are available in the office or clinic. If seen within a few hours the rolled skin should never be sacrificed, no matter how devitalized it may appear. After careful cleansing of the hand with soap and water, and no antiseptics, the surgeon should assume the rôle of the plastic surgeon and proceed to apply these principles in "grafting" the denuded area. It is not infrequent that repair of these injuries may require not minutes but *hours*. Final results will more than justify the fastidiousness of the surgeon. Should skin be lost, the surgeon should immediately replace it with free grafts.

The most ideal is the full thickness graft; however, this leads to delayed donor area healing so that the thick split graft may be advisable in some cases. This, however, does compromise the ideal

full thickness graft. The extensive hand injury will require a longer operative time than most major abdominal procedures and justly so, for the permanent disability may be far reaching.

While the wringer-type of hand injury with skin peeling provides the ideal injury for immediate skin grafting, similar injuries, such as result from automobile accidents, with extensive soft tissue and deep structure injuries, lend themselves to immediate grafting for skin losses. Here again basic cleansing principles must be adhered to before repair may be instituted.

Any surgeon who presumes to accept responsibility for the care of these injuries must be adept in nerve, tendon and bone surgery, and must, therefore, keep himself familiar with current advances in these particular fields. It should be quite evident that a neurosurgeon, an orthopedist and a plastic surgeon would be a crowd in the operating room. It should be emphasized that even in these ragged, lacerating injuries, radical débridement should be frowned upon; every available milometer of skin should be saved, even at the expense of long operating time. By careful fitting and key suturing many denuded areas may be covered. Palmar surface skin is frequently lost because of failure to realize the increased thickness of the skin in this area so that sutures are placed superficially. After the slough of the thick cornified area, the suture may not even have penetrated the thickness of the skin, hence, lack of co-aptation and resultant poor feeder circulation. Denuded tendons may be grafted with confidence of success, but should have full thickness grafts and careful attention to proper pressure dressings.

The interposing of fat, or the cutting of the graft with fat to prevent adhesions between the graft and the tendons, is an excellent method to insure its death except for pedicle flaps. Fat does not prevent adhesions; on the other hand, it is an excellent insulating tissue against good blood supply to the base.

The use of mechanics waste as a pressure medium, as popularized by James Barrett Brown, is infinitely satisfactory, provided the dressing is applied carefully so that it will function. It certainly has no magic inherent powers to apply uniform pressure unless equally distributed pressure is applied through the application of the dressing. Once the dressing has been applied and examined daily to determine whether it is functioning properly, it should be left alone for a week. Frequent dressings only increase the hazard of infections; the gentlest change is traumatic to delicate healing structures. Curiosity to view grafts should be repressed.

It is essential that these injured hands be splinted in complete rest. Many a two-hour painstaking operation is ruined in a few minutes by the patient struggling in postanesthesia. If proper, adequate, light splinting material is not satisfactory, plaster should be used which later may be shelled for observation and dressings.

The surgeon who cares for these hand injuries should acquaint himself with the armamentarium of the plastic surgeon, so that he may have adequate and applicable instruments, otherwise he may find himself a boilermaker in a watchmaker's shop. Careful, gentle and meticulous handling of tissues, to a point of being ultrafastidious should be the guide posts of any surgeon in the care of hand injuries. Time taken in the care of these injuries should be of no importance.

Some time has been consumed in detailing the hand cleansing as it is so vital if grafting is necessary. As a matter of fact, the method of cleansing of the hand may quite definitely determine success or failure of the graft regardless of how perfect the graft may be cut and applied.

Full thickness skin loss in burns lend themselves admirably to early grafting and here again, basic cleansing principles apply possibly more forcibly than in lacerated wounds. After all, a burn is a wide open wound and should be treated as such. As soon as a clean base is established split grafts should be applied. One thorough and careful operative procedure at this time may save numerous later operations which frequently are basically palliative. The dermatone makes available an accurate device for skin splitting so that there is no excuse for delay in grafting. It has been pointed out in treatment of burns, even if the graft is a failure, it has supplied an ideal dressing and to some extent has prevented contracture.

The potentialities of permanent disability with economic loss in injuries of the hands may far surpass potentialities in so-called major surgery elsewhere.

Treatment of injuries of the hand demands far more consideration, far more sober thought to combat permanent disabilities. These injuries are worth most considerate, thorough and gentle care and should be elevated in importance to the sphere of major surgery. They should also be recipients of the fruits of advances in specialized lines of surgery. Possibly it is unfortunate that the largest percentage of them are destined to be cared for by the general surgeon, but with wisdom this garden variety of injuries may be the vehicle to carry him where he may do many things well.

DISCUSSION

EARL C. PADGETT (Kansas City, Mo.): Dr. Hawkins has, we all agree, brought up a subject that is very timely. He has tried to lay down general rules for the care of hand injuries and as in all cases in which one lays down general rules, there are many exceptions which one might use with advantage. I will not go over those particular things, only to say that sometimes I would think that possibly fractures might not be of secondary importance.

Although one might lay down a general rule that general anesthesia is always necessary, I think that possibly one sees many cases in which local anesthesia might be used with some good judgment.

I have heard many men state that the completely severed portion of a finger should be reattached and given blood supply, but I have never seen such a thing happen if the cut was deeper than the subcutaneous tissue.

The statement was made that the most ideal graft is a full thickness skin graft. Personally, we have not used the full thickness skin graft except on web fingers in children for many years, the reason being that with full thickness skin graft, although the take is perfect and the protection is the best that any skin graft will give, you run about a 20 per cent chance of not getting a take, even on a clean surface, and if you do get a take you may get some blisters. Those do not make so much difference on the hand because one is not looking for an ideal cosmetic result, particularly, anyway. We have used what we have termed (and the term is probably bad) the three-quarter thickness skin graft, basing that designation on the fact that the graft is cut to the last quarter thickness of the skin. This graft gives good protection and the cosmetic result is good and it practically always takes.

On an aseptic field with proper fixation, dressings and so on, one can rest assured that his takes will run close to 100 per cent. Of course, on the granulating areas we do not try to use a fixed skin graft because of the liability that one may lose the graft. We usually cut the skin graft fairly thin, preferring to make the chance of getting recoverage high, and if there is contraction after the hand is healed, one can relieve the contraction.

To generalize then, on the hand we have laid down certain rules which to us seem somewhat of value: For most skin and subcutaneous tissue losses or contractions in the palm or over the dorsum of the hand or of the fingers, the application of the proper skin graft is preferable to the application of a skin flap. The exceptions to this statement are met when tendon, bone or joint is laid bare or when it is anticipated that a tendon grafting operation is to follow. Then a skin flap is definitely the indication.

On the fingers on which it is essential to cover a guillotine amputation or the original injury has been of the guillotine type, to get added length with adequate protection a pedicle skin flap is the indication. This is also the case when one side of the finger has been denuded down to the bone. When it is desirable to increase the length of an amputated digit, coverage with a skin flap will give one about one-quarter of an inch increase in length in

addition to giving a well padded protection. In my experience, a skin graft does not give sufficient protection over the end of the digit if applied immediately on the granulations springing from severed bone.

Correction of webbed fingers of either the congenital or acquired type is most efficiently done by application of thick skin graft between the fingers after complete severance of the web.

WILLIAM E. BROWNE (Boston, Mass.): I do not believe I have a single thing to add to Dr. Hawkins' paper. There is nothing we do that is materially different from what he has outlined in his paper. Perhaps we would not go quite as far as he goes in saying that fractures are not of prime importance in changing abnormalities in the skeletal framework of the hand before attempting the soft part repair. If, for example, you consider what has been said this morning about subtrochanteric fracture, about the vastus lateralis, the gluteus maximus and adductor magnus, and the danger of injury, and if you transfer that to the hand, to the thumb, for example, and the structures attached to the outer border of the proximal phalanges, that is the outer head of the phalanges and the inner border, the inner head of the flexor pollicis proximal and the oblique adductor of the thumb, to attempt a skin graft between the first and second fingers with those antagonistic muscles unattended to by lines of fractures not repaired, would result in considerable deformity as one might attempt to reconstruct the hand with soft part repair of one kind or another.

I do think that Dr. Hawkins' note on anesthesia, passed over lightly, is of importance. He has underlined in his paper, which he was kind enough to send me, the words that we never use local infiltrative anesthesia in attempting these things where other forms of anesthesia may be permitted. I do think it is unfortunate, when one sees these grafts which do not hold and when one sees in the fifth or sixth layer of the skin (removing sutures ten days rather than a week after they have been put in) that the deeper layers have not been attended to. One can avoid that very nicely, obviously, by undercutting the edges properly before grafts are put in.

I have not had the nice results with Dr. Padgett's dermatone, which everyone should have, and I have used it in other parts of the body with better results sometimes than in the hand; as I say, I have not had the results which I am sure he has had. I do think that it is important in these finger injuries, where the tips of the fingers are gone, in using little postage stamp grafts there, that thick skin grafts are advisable. I am sure it is almost impossible to talk to men who have had tremendous experience in this field, to express certain dogmatic points, except to prevent those who are not adept in handling these things from attempting to handle them. They must be done, obviously, by persons who have had considerable experience.

I think it is most important to bear in mind what Dr. Hawkins said about not wasting ruffled up and raveled up plaques of skin. Suppose the skin does not take at all which one unruffles or pulls down; there is much

better healing where one uses that skin, even though a good part of it is sacrificed later on, and in the event that full thickness graft or some other form of graft is later necessary, it will take very nicely. I think in a certain percentage of these cases—and I cannot give any figures—in which the tip of the finger is done, that it is highly desirable, although there is a good deal of atrophic disturbance, to inject the digital nerves before a graft is placed or an inlay is laid, so that the tip of the finger, when one attempts to use it later on, will not be very painful.

I heard Dr. Lund, who was my great teacher, define minor surgery one time as surgery done by minor surgeons, and the more minor the surgeon, the more major became the surgical thing he was doing. If that is true, it certainly is true in the hands, and I would advise persons going to the other end of the ladder, to learn how to do a thyroid, to know the triangles of the neck, to be able to do a breast very thoroughly, to be able to take out a gallbladder well, to do a hernia very, very nicely, and after that, come back to the hand which seems to offer so many difficulties.

Lastly, I might be permitted to pay tribute to the President of this Association, of which I am not a member, for the very excellent work he does here in Boston on these very difficult problems involving crippled hands.

A. William Reggio (Boston, Mass.): I would just like to add one word on a thing that we are apt to forget in these hand cases. We speak of splinting the hands right along, but we must not forget to splint those hands in the position of rest, sometimes called the position of grasp. With relaxation, it avoids tension and is infinitely more comfortable, so that the position of grasp or relaxation, if possible, is the way that we should do our splinting every time.

In the second place, do not forget the shoulder. So many times we see these people come in who have had trouble with their hands, and at the hand clinic at the Massachusetts General Hospital, we have the habit of saying to these fellows, "Put up your arms," and there you are. The injured hand gets a glued up shoulder. Always give the instruction, on whatever you do on the hand, that the patient must put that hand up over his head at least three or four times every day. That is a point worth remembering.

Joseph L. Burnett (Boston, Mass.): There is one thing in this discussion of hands that has not been mentioned and I think it is very important. Most of these patients with injured hands land in the hospital and the suturing of tendons and other things connected with compound injury of the hands are done usually by the lowliest intern on the staff. I think you men should go on record as saying that all hand injuries other than a simple lacerated wound, should be done by no one less than the resident.

Thomas L. Hawkins (closing): I certainly did not wish to convey the impression that the fractures were unimportant. They certainly are important and it does not do you very much good to spend a lot of time on the

soft tissue and then not take care of the fracture. On the other hand, I still believe that in the treatment of these, you may get a perfect result from a fracture standpoint, and then have a nice infection which will obviate all of your good work on the fracture.

I think we ought to remember what Dr. MacFarlane said about the use of sulfanilamide. Certainly, I think it is a grand adjunct to surgery, but just like antiseptics, it is not the really important thing. The really important thing is the careful cleansing, the débridement of the wound, and we certainly should not expect miraculous results from the use of sulfanilamide.

Remember what I said about not dumping large amounts of sulfanilamide into the wound.

Dr. Reggio's statement on physiological rest is of a great deal of importance.

INTRACAPSULAR FRACTURE OF THE NECK OF THE FEMUR TREATED BY INTERNAL FIXATION

AN ANALYSIS OF 128 CASES

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THIS is a report of 128 consecutive cases of intracapsular fracture of the neck of the femur in which the patients were treated by one method. It comprises all but seven cases having such fractures, which either of us have seen in the seven years covered by the report. The seven cases in which pinning was not used were patients who refused operation or the patient was obviously in a dying condition when first brought to our attention. Nevertheless, among those patients operated upon, tremendous risks have often been taken on very feeble patients. Many times we have been gratified to see an individual, who at first appeared to be headed for the grave, walk out of the hospital on his own feet. Our deaths occurring in patients while under treatment have been very few, and no patient has died within forty-eight hours after operation.

PROCEDURE

The operation has been described in previous papers and only a few points need be mentioned. In our early cases, the pins were introduced in a converging manner as first described by Austin Moore. We later got away from this idea, and for approximately the last 110 cases have tried to get our pins in as nearly parallel as possible. We have also attempted to start the pins at a lower point on the shaft than we did in the beginning. For this reason, we have sometimes had to drill holes through the cortex of the shaft. There has been one other change in the operation and that is, instead of spinning the pins in, they have been driven in. We have devised an instrument consisting of a rod of steel into one of which the pin is screwed. This rod then acts as a handle, and with an ordinary hammer, the pin is driven home.

The results of this series are very gratifying. Only nine patients or 7 per cent have died while under treatment in the hospital. Chart I lists these deaths and gives the cause in greater detail.

The individuals operated upon in this series were nearly all quite elderly and many very feeble. The average age was 67.3 years, the youngest being thirty-five and the oldest eighty-nine. There were, however, five patients under forty years of age. It is interesting to note that three of these were patients in the Insane Asylum, the fracture occurring while under metrazol treatment. The fourth was injured in some kind of drunken brawl, the nature of which he cannot explain, and the fifth had many other fractures. Almost four out of five of our patients were females. The right hip was broken about as often as the left.

RESULTS

Anatomical Results. Fifty-nine cases have resulted in firm bony union proved by *x*-ray and rated by competent roentgenologists. Nine cases were rated as union forming on last *x*-rays available. Whether they have or will go on to firm union or not, we do not know as no further check has been made in this regard. Several are fairly recent and the others have been lost sight of. Twenty-six cases have been rated by us as clinically united, but in which no late *x*-ray is available. In this group, however, function is excellent or good in every instance. The reason the check-up *x*-ray has not been obtained is, in all instances, because the patient refused to come in for such a procedure, generally with such a remark, "My leg is all right. I don't see why I should have another *x*-ray."

Sixteen cases have resulted in frank nonunion, the details of which are brought out in Chart II. Two cases have been lost sight of and the net result is not known, and seven patients have been operated upon too recently to appraise.

Boiling down these figures, when one removes the deaths and cases lost from sight or too early to appraise, we have a total of 110 cases.

	No.	Per Cent
Firm bony union	59	53.63
Union forming when last *x*-rayed	9	8.18
Excellent clinical results without check-up *x*-ray	26	23.63
Nonunion	16	14.54
Total	110	

Inasmuch as it is not probable that any of those put down as clinically united are really ununited, in view of the function, it is probable that the percentage of nonunion is adequately stated. However, listing those only in which definite *x*-ray evidence is available, the following figures are obtained:

	No.	Per Cent
Known bony union	59	78.66
Known nonunion	16	21.33
Total	75	

These sixteen cases of nonunion, however, bring out some interesting facts when studied more clearly. Chart 11 goes into this in greater detail.

FUNCTIONAL RESULTS

The functional results are as follows:

	No.	Per Cent
Rated as excellent function	55	55
Rated as good function	24	24
Rated as fair function	12	12
Rated as poor function	9	9
Total	100	

There are several others which are not rated at all, either the patients died, the operations were done too recently or a mental condition or another disease has become superimposed upon the hip fracture, and the patient either cannot or will not even try to walk. Several such patients are known to have good bony union with perfect anatomical position. It might be assumed that the function would be excellent in these latter cases if the patients would or could walk, but we thought it better not to score these from a functional point at all.

It is interesting to note in this connection that some of our cases of known nonunion function well, whereas in some of our cases of known union, the function is only fair or even poor. One of these deserves particular comment:

This is Case No. 23, a woman forty-three years of age, who received a transverse central fracture of the neck of the femur in May, 1938. She was pinned on the day following the accident, was out of bed in five days and

home from the hospital in fourteen. For some fifteen months she did very well, walked without pain, limp or any loss of function whatsoever. However, in the late summer of 1939 she began to develop pain in the hip and went to a surgeon in another city who believed and told her that one of the pins was too long and should be removed. She submitted to this operation, having all three pins removed, but felt no better. Her leg was strong and there was no shortening, but there was considerable pain. She finally appealed to me almost four years after the original operation with what she thought to be several inches of shortening, great limp and considerable pain. The *x*-ray shows a dead head, which has worn off to such a degree that abduction is impossible, and although there is no real shortening in this leg, the apparent shortening, because of this adduction deformity, is about 3 inches. This case represents one of four known dead heads in this series. The other three ended in nonunion. This one, however, united.

There is another factor in these cases which is quite significant and that is the time element. The majority of patients were operated upon promptly. Three times the operation was done on the day of injury. All three of these patients did well. More frequently the operation was performed on the next or the following day. However, sometimes we have had to wait because of intercurrent conditions, in one instance as much as thirty days. This latter case also did well. In two cases which were done approximately sixty and seventy days, respectively, following the injury, nonunion occurred, one of which went on to union following a Schantz osteotomy. The length of time patients have remained in bed has usually been short. At least three patients have walked on their legs (without permission) on the day of operation, and all three of these went on to good union. Many patients have been out of bed within three or four days and unless some intercurrent condition was superimposed, the average stay in bed was less than a week. Many of our patients bore some weight within less than a week. It is true that in many of these patients, the neck shortened a little, but it is equally true that in some of these the neck did not shorten. It is also true that in many of those who remained in bed for months, the neck sometimes shortened or sometimes did not shorten. We believe that shortening of the neck is due to interference with the blood supply and not to early weight bearing; and that if it is going to occur (and one can never know this for certain), weight bearing on properly applied pins is probably an advantage.

CONCLUSIONS

We wish to present a method of internal fixation of fracture of the neck of the femur, which in our hands has been found satisfactory.

One must use good materials, and these materials must be introduced into a femur, the fracture of which has been accurately reduced, and so introduced that the fragments are held so firmly together that no other splinting is necessary. While our records of cases in which the result is accurately known show 78.66 per cent of

NONUNION

Case No.	Age	Cause of Nonunion	Remarks
1	45	Gross infection	Has fibrous union with good function, about ¾ inch shortening; walks well
2	44	Dead head	Was first in a cast, complaining bitterly of pain. Then pinned on tenth day after accident. Continued to complain bitterly of pain. Pins became loose in six weeks and patient was repinned. Continued to complain bitterly of pain for several months. Ended up with complete necrosis of the head, about 1½ inches shortening; walks fairly well and tends to business as merchant
6	61	Dead head	Patient got along fairly well for several months and was walking. Gradually began to have pain. Pins loosened up and *x*-ray showed a dead head with upward slipping of the lower fragment. Patient had good function. Had pain for about one year which gradually subsided. Returned four years later; died of malignancy of kidney; was walking well at that time
9	70	Failure of bones to grow	Pins became quite loose; patient already badly crippled with arthritis
12	51	Dead head	Patient complained bitterly of pain for about two months after pinning; walks well now but has considerable limp and considerable shortening
51	35	Insane patient	Insane patient; this was a metrazol case; patient already confined to insane asylum and still there; no co-operation
58	89	Failure of bones to grow	Patient was very feeble, walked on the pins for about a year. Pins then became loose and fracture fell apart. Was repinned but died a few months after this without ever gaining union; function for the first year very good
60		Insane patient	Metrazol case; had pneumonia; still confined to insane asylum
63	40	Rheumatoid arthritis	This confined her to bed for months before the fracture; complete nonunion; is still in bed but has no pain
77	75	Pinned too late following injury	Injury had occurred many weeks before, impossible to know how long, as the original injury was probably an impacted one and the second injury of at least one month before, breaking the impaction
87	67	Pinned too late following injury	Patient pinned at least ten weeks after injury, preceding which there had been no treatment; patient got union with Schantz osteotomy
91		Insane patient	No co-operation
95	85	Improper introduction of pins	*X*-ray broke down at the time of operation and all pinning was done blindly and was not done well
105		Insane patient	No co-operation
112	80	No doubt due to multiple injuries following pinning	Patient became delirious, fell out of bed several times, had to be shackled, then jerked and pulled the shackles continuously; died in about three months of heart condition
113	83	Failure of bones to grow	Patient returned one month after pinning with thrombosis of popliteal and gangrene of leg; did not survive amputation but it was apparent from condition of hip that union would not occur

union, we believe that this figure is really entirely too low. There are several of our cases of nonunion in which union was not even expected; two were definitely done too late. Other cases have failed to unite because of conditions beyond our control or what in view of our present knowledge appears to have been an inefficient application of the method. We firmly believe that when one is confronted with a new case, properly selected, it can be assumed that in at least nine out of ten times union will occur.

DEATHS

Case No.	Age	Postoperative Day of Death	Cause
3	89	3	Cardiorenal
10	89	5	Uremia
19	86	40	Inanition
32	63	16	Coronary
40	79	6	Pneumonia
46	76	8	Coronary
59	73	14	Coronary
72	78	6	Pneumonia
120	72	6	Cerebral accident

DISCUSSION

N. J. GIANNESTRAS (Cincinnati, Ohio): Talking about fractured hips in Boston is like bringing coals to Newcastle. Some of these patients were interesting, particularly the ones we had at the insane asylum. The first individual I had was a "metrazol" fracture, and at the end of six weeks when we took our first check-up plate after the operative plates, I noticed that he had excellent union there and I happened to make that remark in his presence at the time, and he said, "Doctor, do you know how I got a good leg out of it?" I said, "No." He said, "From the second day after the operation, I jumped off the bed onto the floor so my leg would get good and strong." Perhaps you can imagine a man who weighed about 160 pounds jumping off the bed, about three feet high, onto a concrete floor and those pins holding. They seemed to do the work.

There is another interesting thing about this procedure, and that is the possibility of getting them up much earlier. Dr. Carothers had a case of a diaphragmatic hernia in which he was told that he would have to pin that

hip right away because if that woman did not get up, she would not be able to eliminate properly because of the diaphragmatic hernia, and that she would get in trouble. I believe that she was able to sit up and was up in about three or four hours after the operation.

Now, as Dr. Carothers mentioned, there is nothing new about pinning but it is simply the method of fixation. We have seen cases in which the neck has shortened but because the pins are parallel, they have either gone into the acetabulum for a slight distance, and then even though in some instances the pins have been impinging upon the acetabulum, it does not seem to make any particular difference and does not seem to cause any particular pain in that individual. In other instances, pain has been induced by impingement of the pins in the acetabulum and they have been removed.

Another observation is that, of late, they have found that it is sometimes a good idea to place the head in a little valgus position in comparison to the neck so that when the patients are up and around, they get a more definite thrust rather than a shearing stress at the point of fracture, and thus probably help in union.

E. Payne Palmer (Phoenix, Ariz.): This excellent paper and the splendid discussions leave very little to be said regarding the fracture itself. The medical profession is derelict in its duty when it does not explain to a patient who has a fracture of the neck of the femur that a large percentage of these patients are going to have crippling, that the percentage of satisfactory results has been increased by the various methods that are being used but we still have this percentage which is appalling, and as Dr. Speed says, this is still an unsolved problem.

So if this organization could do anything toward directing the profession as a whole to tell these people, when they get a fracture of the femur, and to tell the family, that there is going to be a percentage of failure which they have to anticipate, it would do a lot to unload the responsibilities of the man who treats fractures of the neck of the femur. Please remember this.

Captain Boardman M. Bosworth (New York, N. Y.): I hesitate to impose upon you by taking the floor, and my only excuse is that in a way I represent another man. I am speaking for Dr. Preston Wad, a member of this organization, without his foreknowledge and consent, so I hope my remarks will not be taken too seriously.

There are just two or three questions that I wish to put to Dr. Carothers. The first is, do you use three pins all the time, or do you sometimes use four? I ask that particularly because we have had the sad experience in two cases of having the pins break within the first six months or a year, and in one of those cases the distal half of the pin we found located right in front of the bladder when we went to remove it. I wonder whether you have had the experience of pins breaking, and also whether you have had any of your pins wander out the other way, in other words, extrude themselves.

I also did not understand exactly how long your average follow-up was. At the City Hospital where Dr. Wade and I are on the staff, on Dr. Ban-

croft's staff, for the past five or six years we have had the privilege of handling all the fractured necks of the femur that came in on Dr. Bancroft's service, and as a matter of fact we are right in the middle of preparing a paper to give the follow-up results on 200 cases of fractured necks of the femur, both intracapsular and intratrochanteric, which we had nailed during that time. Unfortunately, that paper had to be postponed, but these questions arise as a result of our experience in that work.

I would like very much to have Dr. Carothers say whether he has a definite time at which he urges his patients not only to get out of bed, but to bear weight, first with crutches, and secondly at what time he likes to have them begin complete weight-bearing. Our policy has been what I presume his is, to get the patients out of bed and active as soon as possible. In other words, we have our patients, on the average, out of bed either the next day following operation, or the second day, with very few exceptions, and we have them up either on crutches or on a creeper within the first ten days to two weeks.

Then in the technic, I was very much interested in the angle iron with the holes in it that was shown. That struck me as a very clever arrangement. It had not occurred to us. We have been using two pennies, one of which we apply with adhesive over the major portion of Poupart's ligament. The other we place over the femur where we think we are going to put our pins in, and then we take our *x*-ray before operation, and those two penny marks are fixed with gentian violet or similar dye before the patient is prepared. We use those as guides very satisfactorily.

Finally, I may say that we, too, have had some experience with insane patients. Both of us have taken trips out to Kings Park Hospital on Long Island to nail hips which have been fractured by metrazol.

KENNETH M. LEWIS (New York, N. Y.): I would like to confirm an observation that Dr. Carothers made. At Bellevue we have been using Smith-Petersen nails. We have tried to get ambulatory Thomas splints for our patients to walk in, and we have always tried to avoid early weight-bearing, apropos of Smith-Petersen's dictum that when they do bear weight, there is a shearing force and a compression force between the fragments. Unfortunately, we cannot get ambulatory splints for all of them and they are put on crutches, and we have had a fair number of them—I should say fifteen or twenty of them—who simply would not use their crutches. They would be gotten out of bed and for the first few days would be in a wheel chair, but they were walking at the end of possibly a week or ten days, and I would come on the wards and see some of these people with their crutches over their shoulders, parading up and down the wards.

When the first few were observed, I was sure something was going to go wrong, but surprisingly enough, every one of those cases got union, and some of them got union very rapidly. This is the first observation I have ever heard made confirming the findings that we have had in a few cases. Why they get union any more rapidly than the others, I do not know. Ac-

cording to Smith-Petersen's idea of a shearing force, that should interfere with union, but in these cases of ours we have had the same experience that Dr. Carothers has had.

H. Gurth Pretty (Montreal, Canada): Dr. Carothers' paper has interested me very much, and particularly the fact that he has made reference to his follow-up cases in a period of five or six years. We have been reviewing some of ours in the last two or three months, and in the follow-up there are two points that I think are worth mentioning: First of all, in a group of about seventy-five cases, using a single pin, we have had four cases that have developed an adventitious bursa over the head of the nail, and that developed in a period of about eight months or a year; and by excising the bursa or extracting the nail at about eighteen months' time, our difficulties ceased.

On reviewing several of our cases which have gone a period of six years, we have been surprised to find that in three of them, they are developing a small triangular aseptic necrosis of the head at the apex of the nail. The union is perfect, the lines of stress and force are perfect, and in only one case did this individual complain of a suggestion of pain. The others had a slight click. But the interesting thing is that at the apex of the nail, in two cases in which the nail had been extracted, one in which it had not, there was this little triangular area of aseptic necrosis.

In checking up a group of old cases that had been treated by the Whitman method, or modifications of plaster, and looking over those, over a period of six and eight years we found that some of these had a similar condition but instead of occurring at the apex of the nail, it was occurring on the interior border of the neck, or rather the head.

I merely bring this up as an interesting point because we are interested to see what is going to happen and it may come to the point that we may have to do a reconstruction operation sometime in the future or put in some of the vitallium cups.

William Darrach (New York, N. Y.): I think it is hard for anybody to keep quiet on hips. I think it is still an unsettled problem. We are struggling to find an answer and we are still in doubt about a good many details. You think you get an idea along one line, and then you talk to somebody else and he brings in some more facts and things and it unsettles you.

I have been distinctly unsettled by the discussion this morning, and I am beginning to wonder about two points. I think it is quite outstanding that the author has had only one flat head in a series as large as that. We certainly can beat him on that because we have had a great many more flat heads develop during the second, third and fourth years, and because of that, I am wondering whether the three or four pins may not be a better bet than the Smith-Petersen pin which we have been using.

The other disturbing factor to me is the early weight-bearing. We have been fairly convinced that the percentage of flat heads, with or without bony union, is greater if the cases start weight-bearing early. I do not know

whether we are right or wrong, and as the figures are coming in from a good many places where early weight-bearing has been followed out, we are beginning to wobble in our position that at least six months should be allowed before any weight-bearing is tried. That, I do not know.

There is one thing I do know though, and that is we are delighted with the improvement in our results since we gave up using caliper splints in the upper treatment. The more I think about the caliper splint, the more ideal I think it is as one method of destroying any union that may be starting. The ring of the splint makes a beautiful fulcrum right over the site of fracture, not only for flexion, extension and abduction but rotation, and I think that our earlier experience using the caliper splint was perhaps one reason why our earlier results were not as good as they should be, and were not as good as they have been more recently since we gave up the caliper splint.

Of course, no matter how firmly you lay down the rule to these people that they must not bear weight, a great many of them do, so we are sneaking in a little series of early weight-bearings against our advice, but it is going to take another good many years to be able to tell what is the main reason for the late changes that come and disturb us and give painful hips, becoming more and more difficult and painful as the years go on.

I got another jounce from Dr. Gordon, and that is that perhaps the biochemist is going to help us. He spoke of these old ladies whose spines begin to decalcify and they get more and more kyphotic and compress two or three vertebrae, and he pointed out the similarity between those and the old ladies who break their hips, and the decalcification there and the later troubles in the heads may be in the same group. I think if they can help us in those cases of back injuries, they may help us on the hips.

Just to sum up, I have no firm convictions today, any more than I had thirteen years ago when I began to nail hips, but I think it is an interesting problem and I think we are learning something every year. I think Dr. Carothers has contributed a very real something with this series which he is reporting.

CHARLES S. VENABLE (San Antonio, Texas): There is one thought that I would like to inject into this discussion; that is, postoperative posture in avoiding adduction. The point of complete rest of the neck of the femur is with the inner condyle of the femur in a line with the superior spine. That is a neutral point. When those patients are permitted to drag the leg in adduction, you are putting a fulcrum right on the top of the fracture, and if they keep doing that, they may work the nail or the pins, or whatever you have, out. I have seen it occur. I have seen two cases in which I had to put a nail in, but the patient would not maintain that reasonable position, so I put a simple hip spike on. I am permitting them to be ambulatory with crutches, with minimum weight-bearing, driving it back because in adduction they do that. I have seen two cases of bent screws or nails just from that.

RALPH G. CAROTHERS (closing): I want to thank the gentlemen very much, and I hope I will answer all the questions.

In regard to early weight-bearing, let me repeat, of all those cases that did well, 80 per cent bore weight early, within a week or ten days. Of those who did badly, only 20 per cent bore weight within that time.

We have attempted in every case to get a little valgus deformity in our reduction. We have not always succeeded but we have tried to do it. We have tried in every case to get the pins very low, and we hope that in these instances we have so pinned the hip that there is not a shearing force but a pressure force; and for those reasons, I believe the neck will shorten sometimes, no matter whether they bear weight or not.

I have one case of a greenstick fracture of the forearm of a child, in which the check *x*-ray, when we finished with it, showed an absolutely hair-line fracture with perfect position. It was put in a plaster cast and six weeks later when the cast was taken off, there was several millimeters' distance between the ends of the bone. The ends of those two fragments had sloughed and there was no weight-bearing, and it was in good firm platers all that time.

I have seen the same thing when transverse fractures have been treated with plates in which the ends of the bone slough and the plate holds the fragments apart, thereby preventing the collapse of the fragments but also preventing union. But in this sort of thing it will not happen.

I cannot agree with Dr. Speed—and I know I am going out on an awful limb to disagree with him—about this early weight-bearing, but we learn from some crazy people. Some of our bad luck came in the insane, but some of our very best luck came with those who bore weight early; and one of the very early good cases that we had was walking right down the hall that same night she was operated upon. That has happened on several occasions and the bones have united, so we began getting them up after that, and getting them up early, and we have had far better luck since we did that. Our bad results are mostly in the beginning.

Now, the biochemist is going to come into this thing some way or other, and I do not know just how, but the first good case I had, and the first good case in this whole series, was the elderly lady that Dr. Giannestras spoke about who had the diaphgramatic hernia and also had pernicious anemia and diabetes. She was getting liver, and I think was getting hydrochloric acid and insulin. Whether they had anything to do with it or not, I do not know, but that old lady lived for four years and I know her hip was good at the end of four years because I got a check picture on it and I talked to her about it.

We have had some pins come out. Some have been loosened up and have extruded. They have been mostly those pins that were put in in too transverse a fashion, too high up, and it is in that converging pin situation where you do get a wiggling like that, but when you have it sliding on it nothing

happens. If you get it up that way, then it does loosen. None have gone into the pelvis, none have broken. And please do not call them wires. That is not a wire. You cannot buy this pin. You have to make your own to make a good one. It is an 18.8 stainless steel that is properly rolled and you cannot break it. Since we got them rolled right, none of them have even bent.

The follow-up has varied from seven years to a few months. Those that have been done within three months have been put down as too early. The weight-bearing has been anywhere from a few hours to never, but in the good cases, it has almost always come within a week or ten days. The average case is gotten out of bed the next day and is bearing weight with a walker, crutches or some sort of thing within a week to ten days. Of course, some people cannot get out of bed. Half a dozen of our cases had strokes when they fell and were paralyzed. Some fell with an attack of angina and their physician made us keep them in bed, and so we could not get them up.

We have used no type of external splinting of any sort whatsoever. We have in our intertrochanterics. That is another story. We have used something to keep them from rolling out.

I do not know why we have had less head flattening. Maybe we will get some more, but we just have not had it; and I do believe that these pins do not chew up the head quite as much as a Smith-Petersen nail does.

ROENTGENOLOGICAL MANIFESTATIONS OF BONE REPAIR

HEALING OF FRACTURES WITHOUT EXTERNAL CALLUS

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THE physiological process of bone repair following fracture is a complicated one.[2] It may be summarized briefly as: (1) Hemorrhage in and about the fracture; (2) decalcification and resorption of devitalized bone; (3) ingrowth of granulation tissue about the fracture; (4) formation of osseous and, to a variable degree, cartilaginous matrix; (5) calcification and organization of this matrix to form bony union, and (6) shaping of the new bone to its final contour.

Roentgenological examination can accurately demonstrate only part of this process,—namely, alterations in the amount and distribution of calcium in the region of the fracture. The other phases of bone repair can, however, be largely inferred by the roentgenological appearance with the understanding that the changes visible on the films are gross and lag considerably behind the histological process. If serial roentgenological examinations are made, keeping in mind the above physiological processes, a decrease in the density of the fracture margins will be seen at about the tenth day. This, we believe, represents the decalcification and resorption of devitalized bone which is a preliminary step in bone repair. After two or three weeks, or sometimes earlier in children, we may see calcification in the matrix surrounding the margins of the fracture which is termed callus. This is the fusiform calcification which we are eager to see and which reassures us that healing is progressing normally. This type of callus is termed external callus.

At the same time a similar process is taking place between the margins of the fracture, uniting the contiguous surfaces. This callus is known as internal or intermediate callus and is not as clearly visible in the roentgenogram as external callus due to the density of the adjacent bone and absence of a soft tissue background. External

callus, on the other hand, is thrown into relief by the surrounding radiolucent soft tissues and can be seen earlier and more clearly than internal callus for this reason.

The roentgenological visualization of external callus is important for the following reasons: (1) Its presence reassures the physician and the patient that healing is taking place. After checking the position of the fragments, the next question of the physician is, Does the *x*-ray show any callus? By this he usually means external callus. (2) The appearance of callus gives some indication of when it is safe to remove splints or other apparatus. (3) It offers a guide as to when the injured part may resume function. (4) The presence and character of external callus is sometimes used to determine the lapse of time since injury. This is frequently important in medicolegal work.

The appearance of external callus is so well known that its further description here is not warranted. Its presence may be comforting, but its absence does not necessarily mean that union will not take place.

An occasional fracture in any location may heal without visible external callus though this is exceptional in the shafts of long bones. The purpose of this paper is to call to attention a certain group of fractures which heal promptly, but do not show external callus formation as a part of the healing process.

When fractures are healing without external callus, it is important to recognize such healing for the following reasons: (1) If it is inferred that union is not taking place simply because no external callus is visible roentgenologically, the physician and patient may become unduly discouraged. (2) If union without external callus is not recognized, splints or other immobilizing apparatus may be employed over an unnecessarily long period of time, thereby increasing atrophy of bone and soft tissue. (3) Medicolegal testimony in which the age of a fracture is estimated by the absence of external callus only, may be fallacious.

One of us (R. G. V.) has previously described the repair of linear fractures of the bones of the cranial vault in which healing occurs without visible external callus.[4]

A recent extensive roentgenological study of phalangeal fractures showed no callus formation about fractures of the terminal tufts although these fractures practically always united.[3] These fractures are very common in industry and they may be clinically well in three weeks but show no union roentgenologically for several months. Thus many days of labor may be lost if the patient is not returned to

work before there is roentgenological evidence of union. This is very important in these days of labor shortage.

It is well known that fractures of the vertebral bodies heal without roentgenological evidence of external callus.

Observation of a large number of fractures led us to believe that fractures involving joints or within joint capsules were also prone to heal without the formation of external callus visible in the roentgenogram. Accordingly, a review of six hundred cases of fracture was made in an attempt to ascertain the presence or absence of external callus in such fractures. Of these six hundred cases, seventy-two were selected for critical study according to the following criteria: (1) Only fractures which lay within or extended to a point within a joint capsule were included. (2) Only cases with examinations at proper intervals to demonstrate callus and with films of a quality that would show a small amount of callus were included.

Obviously a large number of cases were excluded because of heavy casts or other apparatus which obscured the region of the fracture, because a portable machine was used, or as in the case of fractures of the lateral malleoli, only a follow-up examination was made to determine position.

The films of the seventy-two selected cases were studied with particular attention to callus formation. The following points were noted in this study: (1) No external callus was seen at any time about any fracture margin which was within a joint capsule. (2) No external callus was seen in fractures of the tuberosity of the humerus where the tuberosity alone was involved or in fractures of the malleoli below the level of the tibio-astragalar ligament. Frequently, no external callus was seen in the above locations even when the fracture line extended considerably beyond the joint capsule.

In those patients in whom healing took place without external callus the first roentgenological sign of healing was an increase in the density of the fracture line accompanied by a decrease in the sharpness of definition of the fracture margins. This change could be detected after an average period of four weeks which is later than the average appearance of visible external callus.

Further healing, following the initial increase in density consisted of a gradual obliteration of the fracture line with formation of new trabeculas to join those of the adjacent fragments. There is marked variation in the time required for this process ranging from eight weeks to eight months. The degree of separation of the fragments plays a very important rôle; the greater the separation the larger the time necessary for complete healing.

No conclusive explanation is available for the lack of external callus within joint capsules although an inhibitory action has been attributed to synovial fluid.[1] The lack of an explanation does not, however, alter the fact that external callus is inhibited in these locations. In the clinical care of patients with fractures involving joints, it is important to recognize that such fractures can heal without external callus being visible roentgenologically at any time in the course of repair.

CONCLUSIONS

1. Fractures in all locations may heal firmly and promptly without showing external callus visible by roentgenogram.

2. Healing without visible external callus is the rule in those fractures which lie within joint capsules.

3. Healing without visible external callus frequently takes place in fractures which extend into joint capsules even though part of the fracture lies outside of the capsule.

4. It is fallacious to assume that a fracture is of recent origin because visible external callus only is absent.

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DISCUSSION

Philip H. Kreuscher (Chicago, Ill.): The paper by Dr. Vance was interesting to me because of the fact that I have been in error on many occasions, I suppose most of us have, in saying that some of these patients were actually not healed, and we have been in error in not permitting the patients to go back to work or to go back to their usual job in the time when we really should have known that they were healed.

The medicolegal aspects come into the picture. It is difficult to convince a jury, and it is a very difficult thing to convince a Commissioner in an Industrial Commission, an Arbitrator, that a fracture is healed when there is no definite external callus. They have been taught, or at least they think they know, that there must be an external callus before the thing can be healed.

I should like to ask Dr. Vance if he has had any experience with a refracture in some of these cases. In other words, does a refracture take

place more commonly or with less provocation than where there is actually a good deal of external callus present?

Congenital defects, I believe, must be considered in all of these cases, but I should like to make one point and that is, when a patient has one congenital defect, such as a clubfoot or hairlip, or some other congenital defect, look out for some defect also in his spine or some other part of his skeleton. When you have a patient who has a spina bifida occulta or has some other congenital defect in the spine, look out for a congenital malformation of one of the vertebrae also, even an absence of one of the vertebrae.

CHARLES S. VENABLE (San Antonio, Texas): In speaking of Dr. Vance's paper and the question of callus in fractures about the joints, I rather think that it is important for us to recognize that we should not expect callus about spongy bone as we do in diaphyseal bone. We may find a line of union as it goes by the progressive deposit of lime salts in the fracture site, but in the thinner cortex of harder bone about the spongy bone, the process is a different one and it is a much slower process of union.

As you go further into the shaft from a fracture, you see the callus about the fracture in the lower third of the fibula, while you do not see any callus at the malleolus in a femur. I think that is true in fractures of the neck of the femur. We hear it said, "I do not see any callus." I do not think you ever see any callus about the neck of the femur unless it is in malposition. If it is in malposition, you begin to have a pile-up of malleable bone which makes for callus. But in proper position, I think you only see it through the line of fracture in deposit. I think Dr. Vance has done an excellent job in emphasizing that to us in our reading of *x*-rays.

HENRY C. MARBLE (Boston, Mass.): Before you close the discussion, Dr. Vance, I want to ask one question. Dr. Darrach and Dr. Murray in New York have us plating tibias and femurs so perfectly that after they have been plated, the patient gets well and walks around and we never see any callus. Does that fall into the same category?

ROBERT G. VANCE (closing): When I showed you that humerus, I called attention to the fact that any bone anywhere may heal that way. I do not know why.

I wish to thank Dr. Kreuscher and Dr. Venable for their discussion of my paper. In reply to Dr. Kreuscher's question about refracture, we have not seen refractures commonly in that type of fracture in which healing has occurred by internal callus.

SOME OF THE USES OF THE CUTIS GRAFT IN SURGERY*

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IN December, 1941, I read a paper[1] at the Pinehurst meeting of the Southern Surgical Association on some of the uses of the cutis graft in surgery and reported a series of illustrative case reports. Since that time I have had further experience with this method and am making an additional report.

This method was apparently first used by Loewe,[2] later by Rehn,[3] of Germany, and later still by Eitner,[4] of Austria, in plastic and reconstructive surgery. It has apparently been used more generally and frequently in Germany than elsewhere. Rather extensive use has been made of the cutis graft by many of the leading plastic surgeons of this country. Among them are Blair,[5] Brown,[6] Byars,[7] also Davis,[8] Ivy,[9] Fomon,[10] Bames,[11] Straatsma,[12] Smith,[13] Peer[14] and Paddock. Uihlein,[15] after checking over 104 cutis graft cases that underwent operation by Rehn at the University Clinic of Freiburg, was able to trace eighty-four patients. The late results in seventy were good; in fourteen, poor. While this method has been used to a considerable extent in Europe it has not received much attention from general surgeons in the United States.

Rehn[16] and his co-workers, from their experience in animal experimentation, and with the clinical use of the dermal graft, arrived at the conclusion that cutis graft tissue takes on the function and characteristics of the tissue that it replaces; to be more specific, that it may be converted into tissue resembling tendon or that it may assume the characteristics of the various muscular aponeuroses of the abdominal wall.

The usual fate of the cutis graft that has healed in the body tissues is that of a slow conversion into fibrous tissue. Peer[17] and Paddock, in the course of a large series of nasal reconstruction operations, stored pieces of derma along with cartilage beneath the skin for varying lengths of time, from two weeks up to two years. Histological sections were made from these and studied. They found that a gradual process of degeneration of the normal skin structures took

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place, that the sweat glands and the hair follicles were the first to disappear and that a gradual infiltration and replacement by fibrous tissue took place.

FIG. 1. Large incisional hernia previous to operation. (From *Ann. Surg.*, vol. 115, no. 5, May, 1942.)

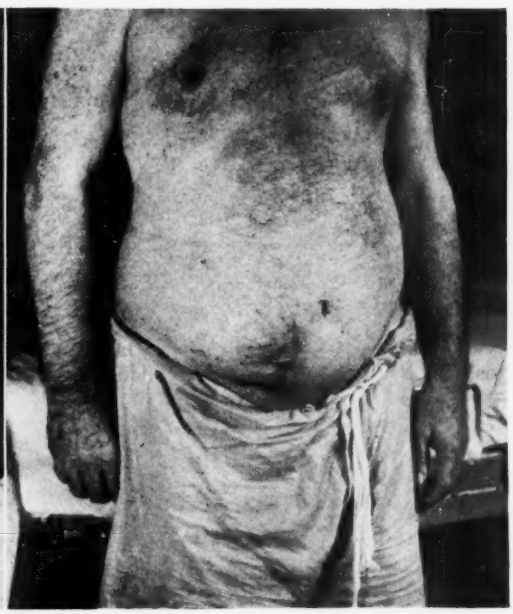

FIG. 2. Hernia ten weeks after operation. (From *Ann. Surg.*, vol. 115, no. 5, May, 1942.)

Cutis graft material can be readily prepared by anyone who is accustomed to the use of the skin graft knife. It has the merit of being easily obtainable at all times and of being more readily available than fascia. It is superior to fascia in many respects. Some have suggested that its use might lead to the formation of epidermoid cysts but the observations by various European surgeons over long periods of time, likewise those of various plastic surgeons in this country, have shown that with proper technic such possibilities are apparently too remote for very serious consideration. It has been definitely proved that the cutis graft gives strong support from the beginning of its placement, that it heals in promptly and that it is gradually converted into fibrous tissue and remains as a definitely firm support wherever it has been placed.

OPERATIVE TECHNIC

The operative procedure for the use of the cutis graft is essentially similar to that used for implantation of fascia. After the defect has

been surgically exposed and repaired as well as possible by suture methods, a graft of suitable shape and size is taken from the antero-external surface of the upper third of the thigh, (although the skin

FIG. 3. Large inguinal hernia.

from other areas may be almost as suitable), the epidermal layer being cut away in a thin sheet with a skin graft razor, after which an area of derma of suitable shape and size is outlined and removed from its bed. A thin layer of subcutaneous fat may or may not be left on the undersurface of the graft. This particular detail apparently makes but little difference. The graft is fixed in place in firm tension with interrupted sutures usually of No. 40 cotton thread. A moderate amount of sulfanilamide, also sulfathiazole, is routinely placed underneath as well as above the graft. Usually not more than 2 to 3 Gm. of each is used in the average incisional hernia repair. Great care is taken to suture the subcutaneous structures in such a manner as to obliterate as nearly as possible all dead space in the region over the graft. No. 70 or No. 80 cotton thread is generally used for this purpose. In many cases, and especially when the graft is large, a bite is taken by the needle about the center of the graft by each individual suture crossing that area.

While the cutis graft transplant is often used as a reinforcement for defective structures, the graft may be satisfactorily used to bridge serious and extensive defects. Where unusual strength is required, a

FIG. 4. Large lower abdominal incisional hernia previous to operation.

double thickness graft can be used with gratifying results. Through-and-through tension sutures are not used in these wound closures. Skin clips are applied, also fine superficial intervening sutures are used to approximate the skin edges. The suturing throughout the wound, likewise ligation of bleeding points, is carried out with due regard to principles laid down by Kocher[18] and later emphasized by Halsted[19] with reference to the use of sutures and ligatures.

When making use of the graft in connection with the repair of inguinal hernias we usually suture the deeper structures to Cooper's ligament, etc. The graft is then sewn in place and covered by the aponeurosis of the external oblique.

Although Rehn[20] advised keeping large hernial repair cases in bed for from six to ten weeks subsequent to operation, we have found this procedure to be quite unnecessary and usually encourage our patients, regardless of the size of the hernial opening, to be out of bed at least twice each day subsequent to the day of operation without untoward results.

The indications for the use of cutis transplants are essentially those for which fascia has been formerly so universally used. More

especially is it indicated for the repair of large hernias of any type in which the openings are so large or the adjacent structures are so weak or defective that a satisfactory repair cannot be made by the usual

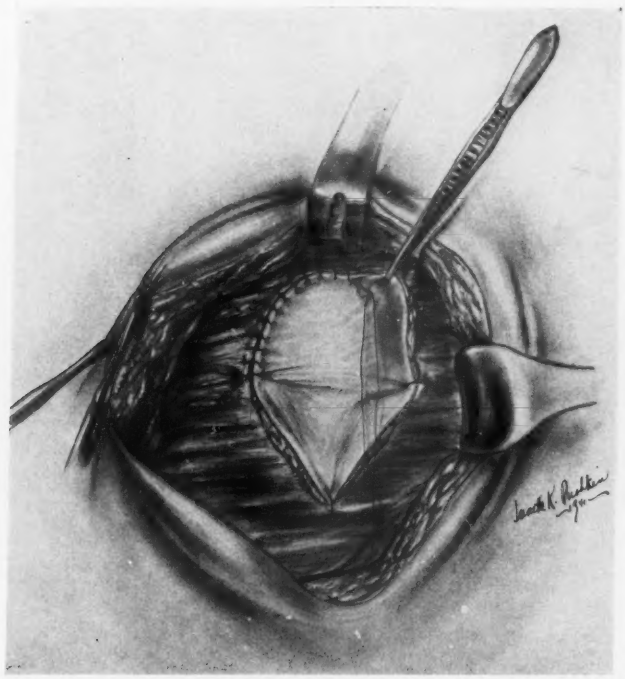

FIG. 5. Cutis graft being sutured to rectus aponeurosis.
(From *Ann. Surg.*, vol. 115, no. 5, May, 1942.)

overlapping methods, joint lining material in arthroplasties, the replacement of damaged dura, the filling out of defects in various parts of the body, more particularly about the head and face, as strong permanent autoplastic suture material wherever it may be needed, the replacement of damaged tendon, the suspension of the uterine cervix in cases of marked uterine prolapse, the reconstruction or reinforcement of weak or absent ligaments, the approximation of bone fragments in the treatment of fractures, either total or stage ligation of large blood vessels.

My surgical associates and I in the Charleston General Hospital have made use of cutis grafts in a total of thirty-seven cases, as follows: Three recurrent epigastric hernias (in one of these a small hematoma was evacuated a few days after operation); seven large incisional hernias of complicated types, not considered satisfactorily operable by the usual methods; seventeen direct and indirect inguinal hernias, which were either recurrent or in which the structures were so defective that satisfactory results could not be expected

by the usual types of repair, (of these last seven were operated by Dr. Bankhead Banks; one by Dr. Hugh Bailey; two by Dr. Victor Skaff, and seven by myself), all with apparent good results. In two

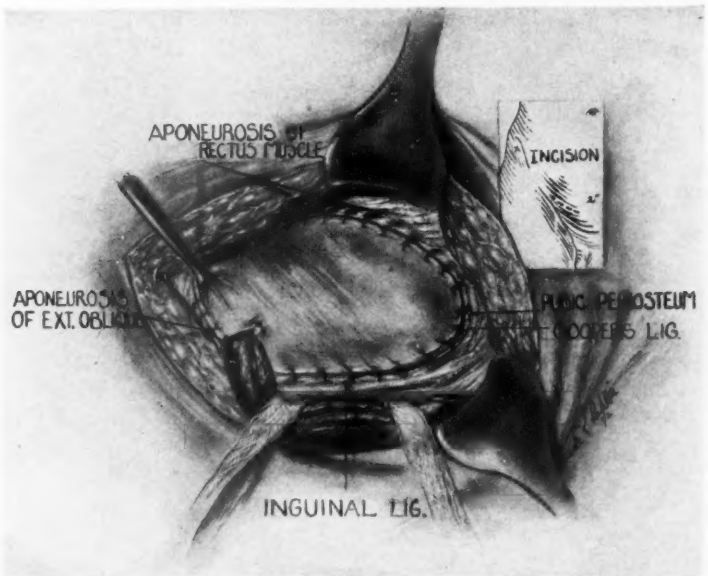

FIG. 6. Cutis graft reinforcement for inguinal hernia with defective structures. (From *Ann. Surg.*, vol. 115, no. 5, May, 1942.)

cases cutis graft material was used to fix the uterine cervix to the undersurface of the rectus aponeurosis just above the pubis for the relief of uterine prolapse; in one case to secure in position an osteo-periosteal graft for non-union of an ulnar fracture; in one case for partial ligation of the femoral artery; in one case of double fracture of the patella; one case for replacement of torn dura; one case of abnormal mobility of the knee associated with defective right lateral ligaments, to support the bowel in two cases of sigmoid colostomy; one case of luxation of the acromial end of the clavicle.

In two of the cases enumerated there were mild infections. In one case a small superficial serum pocket developed but healed in about a week. In another case a moderately sized serum pocket developed in the subcutaneous fat. This persisted until the pocket was laid open after which it rapidly filled in by granulation. In none of the aforementioned cases was there any interference with a satisfactory end result.

CASE REPORTS

CASE 1. B. P., a white male, age fifty-one, was admitted to the hospital May 27, 1940, with a very large epigastric hernia. The bulge on deep expira-

tion was fully 4½ inches across. He had a small ulcerated area over the hernia, apparently due to friction from his clothing. A wire screen was placed over this and the patient was kept in the hospital for three weeks at the end of which time healing had taken place.

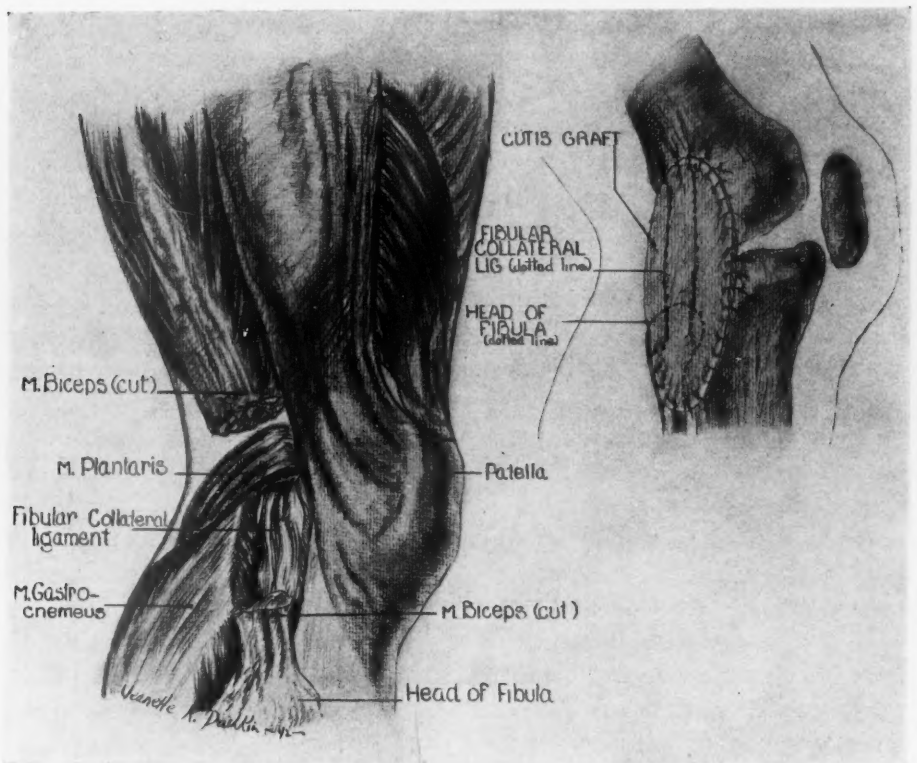

FIG. 7. Anatomy and cutis graft repair for unstable knee due to defective external lateral ligaments.

On July 9, 1940, the hernial sac was excised; the parietal peritoneum was closed by overlapping. The edges of the muscular aponeurosis could not be approximated. A cutis graft from the thigh was sutured over the defect. The patient had an uneventful convalescence. A recent check shows no evidence of recurrence. He returned to his former occupation of coal loading several months ago.

CASE II. T. L. K., a white male, age sixty-nine was admittted to the hospital April 19, 1941, for repair of a large lower right incisional abdominal hernia. He weighed 170 pounds; his blood pressure was 170 systolic, 110 diastolic. The size of the hernial bulge was approximately that of an average baby's head.

At operation on May 8, 1941, the hernial sac was found to contain several loops of small bowel. The opening in the aponeurosis was approximately 4 by 2 inches. This was closed by use of a cutis graft from the

patient's thigh. Up to the time of his death from coronary sclerosis in May, 1942, no sign of a recurrence had been noted.

CASE III. J. H. E., a white male, age fifty-eight, was admitted to the hospital December 30, 1941, with a very extensive incisional hernia in the right upper quadrant, following a gallbladder operation two years previously. There were five separate pockets in the hernial area.

At operation on January 8, 1942, two cutis grafts were used. The first was taken from the redundant tissue in one of the wound flaps and was sutured to the posterior fascia and parietal peritoneum. The remains of the fascia were sutured over this graft. A second cutis graft, from the right thigh, was sutured to the aponeurosis surrounding the hernial opening. The patient was discharged January 16, 1942. To date there have been no signs of recurrence.

CASE IV. H. M., a white woman, age forty-two, height 5 feet 2 inches, weight 284 pounds, with a blood pressure of 220 systolic and 120 diastolic, had a large lower abdominal incisional hernia following cesarean section done elsewhere several years previously.

Operation was performed on May 6, 1942. There was an enormous hernial sac involving mainly the right side of the lower quadrant. When the patient was standing this sac extended within 3 or 4 inches of the knee. After mobilizing the hernial sac and excising most of it, the hernial opening in the abdominal wall was found to be 5½ inches across. By plicating the remains of the hernial sac and by utilizing interrupted sutures it was possible to bring the edges of the aponeurosis in approximation but not to overlap them. The suture line was then reinforced by a running cutis whipstitch for the entire length of the opening. This strip of cutis was taken from the edges of the operative incision.

She was discharged from the hospital May 27, 1942, at which time the incision was apparently well healed. A recent inquiry indicates that this patient remains in good condition.

CASE V. R. B., a white male, age twenty, was admitted to the hospital July 13, 1942, with a compound depressed fracture of the left parietal region. His blood pressure was 130 systolic and 90 diastolic. There was a deep scalp laceration of the left parietal region. *X*-ray examination indicated that a portion of the depressed bone had been driven into the brain for the depth of about 1 inch.

Operation was performed on July 13, 1942, by the surgical resident, Dr. Victor Skaff. The traumatic incision was débrided and elongated. The depressed areas of bone were removed. A considerable amount of brain tissue escaped. The lacerated meningeal artery bled freely and was controlled by electrocoagulation. The area from which the bone was removed was about 1¾ inches in diameter. Since a considerable portion of the dura had been so badly lacerated that it could not be satisfactorily closed, a cutis graft was taken from the left thigh and sutured over the damaged

area of the dura. When in place the graft covered most of the opening in the skull. The temporalis muscle was sutured over the graft and the scalp was closed with interrupted sutures. The patient had an uneventful convalescence and was discharged from the hospital July 24, 1942, apparently in excellent general condition. When seen about six weeks later the area over the bony defect that had been covered by the graft was quite firm to the touch. It was apparent that the cutis graft had in considerable measure formed a protective covering over the area of brain that had been exposed.

CASE VI. L. S., a white woman, age fifty-eight, was admitted to the hospital August 3, 1942. She had a large, lower abdominal incisional hernia that had followed an abdominal operation performed about fifteen years previously. Her blood pressure was 180 systolic and 110 diastolic.

At operation on August 6, 1942, the patient's hernia was approximately 12 inches across. It was found that there were four distinct hernial openings rather closely linked together. The size of the wound after the different sacs had been mobilized was 10½ inches in its greatest diameter. The opening was partially closed by plicating sutures after which a large cutis graft was taken from the redundant skin over the hernial mass on the left side of the abdominal wall and sutured in place. This graft, when sutured in position, covered all four openings and extended about ¼ inch over the aponeurotic edges. The patient was discharged from the hospital August 27, 1942, with the incision well healed. A recent check has not shown any signs of recurrence of the hernia.

CASE VII. W. B. P., a white male, age eighty-four, was admitted to the hospital August 31, 1942. Examination showed a huge dissecting aneurysm of the left thigh. This had developed subsequent to the spontaneous subcutaneous rupture of a popliteal aneurysm of long standing. His thigh was tremendously swollen and the skin over most of it was ecchymotic. A terrific pulsating bruit was present.

At operation on August 9, 1942, a partial closure of the femoral artery with a strip of cutis carried twice around it and anchored with interrupted cotton sutures was made. Pulsation somewhat similar to that of a normal artery of the same size was elicited in the popliteal space for several days subsequent to the partial closure of the lumen of the artery. A slowly progressive dry gangrene, beginning in the great toe gradually ascended the leg. Amputation was suggested but declined. The patient died on August 30, 1942. Autopsy was refused.

CASE VIII. M. C., a white woman, age sixty-three, height 5 feet 6 inches, weighed 175 pounds. Her blood pressure was 170 systolic and 100 diastolic. An enormous right inguinal hernia was present; the sac extended down over the vulva, bordered the right labium minus and nearly reached the knee.

Operation on September 12, 1942, was performed by Dr. R. E. Pence and myself. The hernial sac contained numerous loops of small intestine. The sac, when mobilized, was 12 inches long by 5 inches wide, the dependent portion being the wider. The hernial opening was sufficiently large to admit the male hand (glove size 8½) and arm for general exploration of the abdominal cavity. The hernia was in part of the sliding type, the edge of the bladder peritoneum coming out onto the sac wall. The hernia was repaired as far as possible in the usual manner by suturing. A cutis graft was sutured over the repair. The aponeurosis of the external oblique was sutured over this.

This patient had an apparently normal convalescence. However, on the eighteenth day an abscess became apparent in the more dependent portion of the operative wound. This was opened after which prompt healing took place. So far there is no suggestion of recurrence of the hernia.

Case IX. M. D., a white male, age forty-three, was admitted to the hospital September 22, 1942, with a history of an injury to the right knee received in a mine accident, which resulted in abnormal mobility of the right knee apparently associated with defective right lateral ligaments.

This patient was operated upon jointly on September 25, 1942, by Dr. R. L. Anderson and myself. A longitudinal incision was made on the outer side of the knee so as to expose the head of the fibula, also the external femoral condyle. A strip of cutis of suitable length and width was applied over both of these structures and secured in place with interrupted sutures. Healing was satisfactory but sufficient time has not yet elapsed to determine whether or not the result will be completely satisfactory.

Case X. S. D., a colored male, age twenty-seven, was admitted to the hospital October 30, 1942, with a history of an injury to the left shoulder received in a mine accident, which resulted in inability to move the shoulder in a satisfactory manner. A diagnosis of dislocation of the acromial end of the left clavicle was made.

Under ether anesthesia operation was performed November 11, 1942, by Dr. H. A. Swart and myself. The Bunnell technic was used, a strip of cutis being substituted for fascia lata. Although this patient was apparently in vigorous health, his heart stopped beating and he died in the operating room just after completion of the operation. Autopsy was not obtained.

CONCLUSIONS

Cutis may be used in all cases in which the use of fascia or tendon might be indicated with the expectation of superior results. It heals in rapidly and well; is strong and stable from the time of operation; has great vitality; is able to survive under adverse conditions; possesses great tensile strength; has a good blood supply; gradually assumes the function of the part it replaces; is gradually converted into fibrous tissue and is readily available when needed.

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TRAUMA AND MALIGNANCY

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IN the oldest known record, Imhotep[1] describes in detail forty-eight surgical cases, forty-five of which are due to injury. In Case 39, tumor in the breast, perhaps resulting from injury and in Case 45, benign tumor of the breast, he specifically states it is not due to injury as in Case 39. Since this was written 5,000 years ago, thousands of papers have been published for and against the concept of trauma causing malignancy. Cancer is mentioned in the papyrus Ebers (B.C. 1500). Hippocrates, Celsus and Galen described cancer. Leonides of Alesandria (A.D. 180) recommended radical excision, cutting through healthy tissue with knife and cautery. Lanfranchi of Lyons, in 1290, urged radical operation. Guy de Chaulliac (1300–1386) excised cancer with the knife. Paracelsus (1492–1541) believed that cancer was due to mineral salts in the blood, during which time there was a definite decline in the radical operation for cancer. Bichat,[2] in 1801, distinguished between the stroma as cellular and conceived of its growth by proliferation. Raspail[3] with the microscope, in 1826, showed that the growth of tissue resulted from the multiplication of cells. Schwann,[4] in 1838, established the doctrine of cellular structure as universal principle and discovered the nucleus and nucleolus of the cell. Muller[5] in the same year, published his study of malignant tumors, finding them all to contain nucleus and nucleolus. Virchow,[6] "while admitting the fact that the influence of traumas could not be dogmatically denied, stated that there must be some as yet unknown predisposing factor in the patient and also that the trauma must have been implanted on pathological tissues." Coley,[7] in 1898, reported 170 cases of sarcoma, forty-six of which gave a history of trauma. Ribbert[8] stated that a single trauma may lead to the production of a cancerous growth, Van Bergmann[9] that a single contusion could serve as a predisposing factor in mammary cancer. Ziegler,[10] in 1903, held that 7 to 14 per cent of malignant growths were of traumatic origin. Lowenstein[11] found 241 malignant tumors in soldiers, thirty-nine or 16.5 per cent due to trauma, one malign tumor to 15,000 injuries. Lewy[12] reported thirty-seven tumors in 26,389 injuries from the New York Industrial Commission. Mock

and Ellis,[13] in 1926, reported nine cases which they believed fulfilled the postulates. Villata[14] found 8 per cent of sarcomas and 5 per cent of carcinomas gave a history of single trauma. Mason,[15] in 1929, reported seven cases of carcinoma of the hand and collected twenty cases from the literature due to trauma. Dietrich[16] states if we should regard tumors as arising independent of an anlage or cell nest, we may be free at least to accept the idea partially that the changes in the epithelium and the connective tissue both functional and anatomic can act as a basis for neoplastic changes. Thiem[17] reported 2 per cent of all carcinoma and 5 per cent of all sarcomas as the result of trauma. Loenhard[18] operated upon 500 malignant tumors—477 carcinoma and twenty-three sarcoma—nine of the sarcomas and six carcinomas were due to trauma. Luckow,[19] in 1933, held that there was no dispute on the origin of a sarcoma from trauma. Campbell,[20] in 1941, reported two cases of carcinoma of the upper extremity due to trauma.

ESSENTIAL CRITERIA

Segond,[21] in 1907, Thiem, in 1909, and Lubarsch,[22] in 1912, established the following criteria: (1) The authenticity and adequacy of the trauma; (2) the previous integrity of the wounded part; (3) a reasonable time, relation from three weeks to three years or more in certain cases; (4) continuity of symptoms of the injury with those of the tumor; (5) microscopical or other proof of the existence and histological type of tumor, and (6) the location of the tumor at the point of injury.

Ewing[23] states that the important effects of trauma are: (1) Solution of continuity, minute and gross; (2) separation of cell groups and tissue masses, as of skin, gland and bone; (3) necrosis of tissue; (4) confined hemorrhage requiring absorption or encapsulation; (5) accelerated regenerative processes with hyperemia, and new growth of specific cells, blood vessels and supporting tissue and (6) cicatrization. Rixford[24] reported a case of cancer of the breast in a boy age seventeen, caused by fall on a tree stump striking his right breast. Mastectomy was performed five months later with death four years later from metastasis. When the records were submitted to Ewing he reported, "There is no escaping from the conclusion that in all probability, if not a certainty, the boy presented a genuine case of traumatic mammary cancer." Rienhoff[25] states, "cancer in regard to origin and nature is not an isolated inscrutable problem but one in harmony with microbic-pathological processes in general; it has a

concrete cause, is understandable from the viewpoint of natural science and is explainable in mechanical terms. It is an infectious growth, the same as sarcoma. The cancer cell is a dualistic organism in constitution and action, consisting in a living filtrable virus and a fixed living body cell. A minute investigation of injured single cells and their mode of healing in subcutaneous and superficial situations. Sarcoma is presumed to develop in a comparatively short time after a trauma directly in an injured but still living and recoverable cell to which an invading agent has to get by a circuitous route."

Crile[26] says that "cancer does not occur in normal uninjured cells but occurs after prolonged, repeated injury and repair due to mechanical, chemical, bacterial, electrical or radiation factors. Thus the occasional sarcoma of the thigh which follows a single crushing injury of all the soft parts down to the bone, leaving the skin intact, might be explained as due to autosynthesis. It is significant that this sarcoma sequence occurs only after a heavy blow or extensive crushing. This is comparable to the incidence of sarcoma in bone following a single blow, or in tissues which have not been lacerated and have incurred no infection, there has been no interference with the fullest development of bio-electric currents." Inclan[27] reported eight cases of giant cell tumors of the bone. Case 5 gave a history of a fall on her right knee, after which she complained of swelling, pain and limping. Eight months later the patient fell again on the same knee causing increased swelling and fever and functional disturbance. Three months later the tumor was excised.

Case 6, a white woman, age thirty-five, fell on her left knee, followed by pain, swelling and functional limitation. The pain continued and two years later resection of the tumor of patella was carried out. Gordon-Taylor,[28] in his excellent paper on sarcoma of the muscles and the connective tissue spaces of the limbs, reports twenty cases personally treated and refers to nine seen in consultation. In one case the patient had been struck by a cricket ball on the forearm—sarcoma of extensor muscles of forearm. He also reported one doubtful case of rhabdomyosarcoma of the lateral aspect of the left forearm due to a bite. Leighton and Schmidtke[29] presented before the Western Surgical Association, in 1938, seventy-nine case histories of superficial cancers initiated by a single trauma, which were not biased by any medico-legal phase, concluded that "While no one believes that a trauma in itself produces a cancer, it apparently does set off something, and I believe from our clinical experience that in certain cases, it is the inciting cause or instigation of the cancer."

H. H. Davis,[30] in his discussion concluded, "thus, it is seen that evidence can be given on both sides of the question at least, it can be stated that such an etiology is not common. Trauma reveals more

FIG. 1. Case 1. Roentgenogram showing infiltrating tumor without invasion of bones.

malignant tumors than it causes." Davis, in his excellent paper before this Association, in 1940, is more sceptical, his conclusions in part are: (1) A single injury to previously healthy tissue has never been definitely proved to cause cancer; (2) nobody has succeeded in experimentally producing a series of malignant tumors by trauma; (3) trauma reveals many malignant tumors, whether it can actually cause a malignant tumor is an unsettled point. Metz, in discussing the paper of Davis' reported that in 228,000 accidents in twenty-five years they had seen no case of tumor due to trauma. "In the large volume of negative evidence that can be produced, it would seem conclusive that a single trauma could not produce malignancy, and that this Association could best devote its efforts to aid further in the research on malignancy and help in seeking true cause. Until the true cause is actually found and malignancy can be produced experimentally, we should not hesitate to admit that we do not know the

cause of cancer." In the same discussion, Nilsson reported four cases and Kreuscher two cases due to trauma.

CASE REPORTS

CASE 1. Mrs. E. Z., age sixty-four, in October, 1938, severely traumatized her right hand and forearm in a washing machine wringer. As the

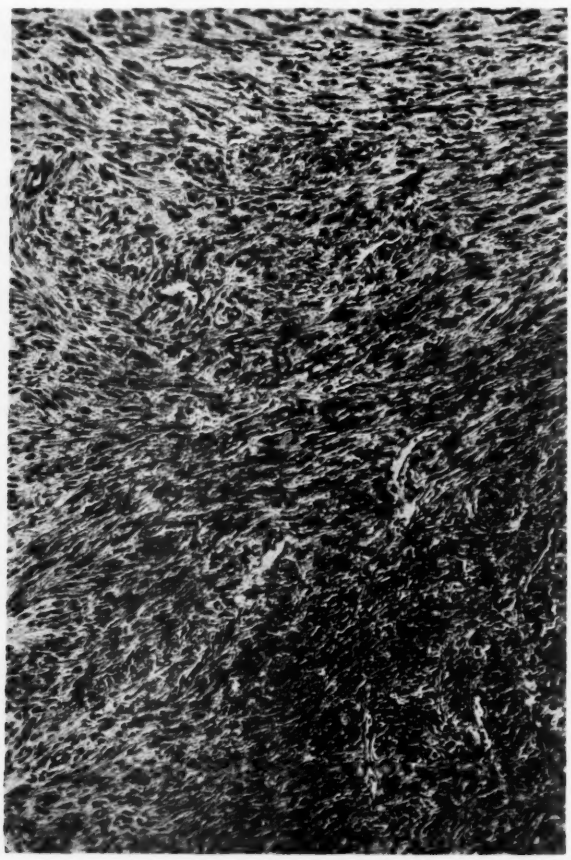

FIG. 2. Case 1. Photomicrograph showing fibrosarcoma arising from tendon sheath.

wound did not heal in eight weeks a skin graft was done by Dr. Werra at the Waukesha Hospital. In January, 1939, she noticed a tumor on the back of her right wrist, which grew to the size of a hen's egg in one month. On February 10, 1939, the tumor was excised by Dr. M. J. Werra. Histological diagnosis by Dr. L. J. Van Hecke: fibrosarcoma. This was followed by six *x*-ray treatments but the tumor recurred in two months. On May 4, 1939, a radical excision was carried out followed by *x*-ray as she would not consent to amputation. A recurrence took place on September 1, 1939. When I first saw her September 7, 1939, there was a scarred area 8 by 10 cm. on the dorsum of the right forearm 3 cm. above the wrist. In this area were

four irregular rounded tumors 2 by 3 cm. in diameter and elevated about 1 cm. covered by atrophic skin adherent to the radius. The patient was admitted to Columbia Hospital September 8, 1939.

FIG. 3. Case II. Roentgenogram showing malignant tumor in distal end of right femur.

Radiographic diagnosis by Dr. S. A. Morton: Chest negative for metastasis, no evidence of bone disease, soft tissue tumor, right forearm on wrist. (Fig. 1.)

Operation on September 9, 1939, consisted of amputation of the right arm 3 cm. above the epicondylus lateralis humeri, which was followed by deep *x*-ray therapy. Histological diagnosis by Dr. G. H. Hansmann: Synovioma, right forearm (fibrosarcoma type). (Fig. 2.) The patient was discharged from the hospital September 27, 1939, in good condition.

On May 27, 1940, her condition was excellent with no evidence of recurrence. On June 10, 1940, metastatic sarcoma of the subclavicular lymph-nodes was found, and on August 13, hydrothorax. Paracentesis of the right chest was carried out four times from August 12 to September 7. The amounts of blood-tinged fluid obtained were 1,550 cc., 1,000 cc., 600 cc. and 1,000 cc., respectively. The patient expired on September 15, 1940. Anatomical diagnosis: Fibrosarcoma of right forearm with metastasis to right axilla, pleura- bilateral; hilar lymph-nodes.

CASE II. Mr. G. V., age sixty, three years ago when getting off a street car in the rain jumped over surface drainage and struck his right femur against a fire hydrant, which was followed by pain in the right knee for three weeks. Ten weeks ago pain recurred in the right knee from stopping sud-

denly while crossing the street; the pain radiated to the hip. He was treated by his family physician. His leg was placed in a plaster of Paris cast for five weeks without relief of pain. When first examined September 28, 1941,

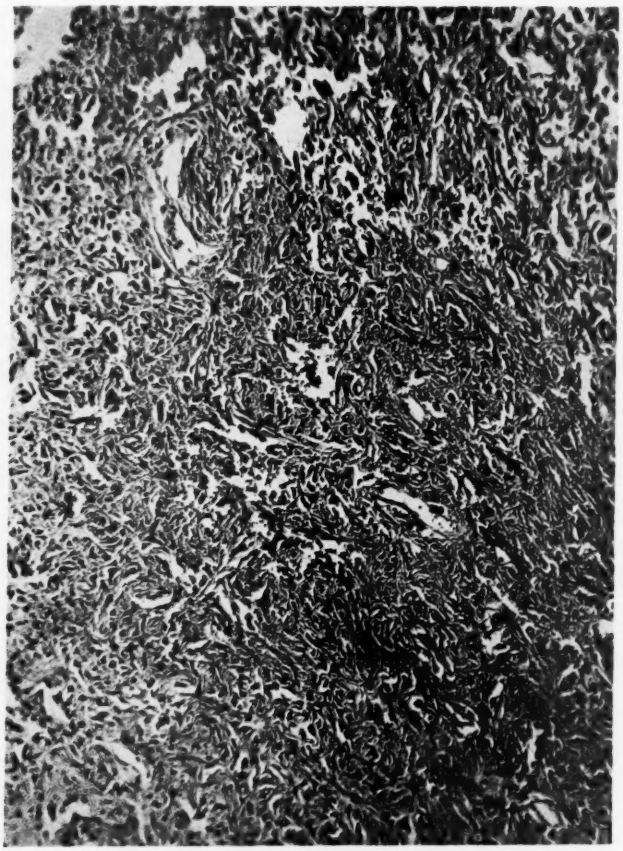

FIG. 4. Case 11. Photomicrograph showing fibrosarcoma of femur.

the skin of the right leg was atrophic, with capillaries dilated. There was pain on light pressure over semilunar cartilages which was increased on abducting the foot. Radiographic diagnosis by Dr. S. A. Morton: Malignant lesion lower end of right femur likely primary bone tumor. (Fig. 3.)

He was admitted to St. Luke's Hospital June 28, 1941. Radiographic examination of the skeleton showed no evidence of metastasis. On June 30, 1941, biopsy revealed fibrosarcoma, confirmed by Dr. Ed. Tharenger. (Fig. 4.) On July 9, 1941, a hip joint disarticulation was performed. He was discharged from the hospital July 26, 1941, in good condition. He refused to take deep *x*-ray therapy; at present there is no evidence of recurrence.

DISCUSSION

From a careful study of the literature it is seen that opinions are divided today as they were a century ago. With the enactment of compensation laws in the various States the medicolegal aspect of

malignancy has aroused a new interest in trauma. In recent years, success has followed efforts to produce tumors under strictly experimental conditions. Both carcinoma and sarcoma have been produced by *x*-ray in man and animals. Marie, Clunet and Baulot-LaPointe.[31] Fibiger[32] produced cancer with sudan III and Yamagiwa[34] and Ichikawa with coal tar. Baggs[35] produced mammary cancer in mice by stagnation of milk. Maud Slye[36] reported 14,000 autopsies performed on mice showing 1,301 indisputable neoplastic growths, of which fifty-one arose at the site of trauma. Pick[37] in studying the records of World War I found two cases of malignancy apparently due to trauma, the age group being an important factor. Behan[38] states, "Heredity, constitutional predisposition and other contributory causes are generally so associated in its origin that the possibility of a single indispensable agent acting as a sole exciting cause is universally doubted." Brosch[39] does not believe a single trauma can cause a tumor while McKendrick[40] states that there is no authentic case in the literature in which it can be proved beyond a doubt that fracture caused sarcoma. Since Schwann discovered the nucleus and nucleolus of the cell the cause and effect of injury to the cell has been studied by surgeons working in every branch of biology. Carrell[41] was the first to isolate in pure strains fibroblasts, osteoblasts, cartilage, epithelium, cancer cells, and leucocytes; and cone[42] cultivated *in vitro* peripheral nerves. Every one who has tried tissue culture *in vitro* appreciates the biological chemical and physical problems involved.

SUMMARY

1. During the past thirty years spent in the surgical clinic and pathology laboratory, two cases of sarcoma which fulfill the postulate of Segond as due to single trauma have been seen.

2. Until the cause of neoplastic diseases has been proved, I will conclude with the words of John Collins Warren,[43] "No young practitioner can be regarded as thoroughly equipped for surgical work who is not both a good Pathologist and an expert Bacteriologist. The confidence born of a knowledge of Pathology and Bacteriology enables him to assume grave responsibilities and to grapple successfully with the most complicated problems. It is from men thus equipped that we have a right to hope that the future masters of surgery are to be evoked."

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MASSIVE INTESTINAL RESECTION

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THE term, "massive intestinal resection," is generally accepted as referring to resections of the intestine measuring 200 cm. (6 feet 7 inches) or more. The mortality in such resection is exceedingly high and, therefore, the fatal cases are not recorded and the successful cases reported in the literature are comparatively few. Our interest in the subject is based upon experience in two cases, one of which was reported before the Western Surgical Association in 1940. There has been a sufficiently long follow-up period in both cases to warrant a statement at this time regarding the merits of the operation.

ANATOMY

Anatomists differ as to the length of the small intestine, Gray giving it as 20 feet 6 inches, Rost as 18 to 26 feet, with an average of 19 feet, and Treves an average of 22 feet 6 inches for the adult male and 23 feet 4 inches for the adult female. Beneke stated that for every 100 cm. (3 feet 3½ inches) of body length there were 387.5 cm. (12 feet 9 inches) of small intestine. The intestine is said to be longer in vegetarians than in meat eaters, as noted in the Russians and Italians who live on a bulky vegetable diet and in the Japanese whose diet consists mainly of rice.

PHYSIOLOGY OF DIGESTION

According to Rost, the small intestine secretes enterokinase which activates trypsin, erepsin which reduces albumoses and peptones to crystalline substances, lipase, nuclease, lactase and carbohydrate. Water is quickly absorbed in the upper part of the small intestine. Carbohydrates are absorbed as monosaccharides, and fats when converted into a soluble form as by the action of fat-splitting enzymes and bile. The absorption of food is practically completed in the small intestine. The jejunum absorbs more fluid and sugar, while the ileum absorbs more proteids and fats. A study of the medical literature indicates that a resection of the small intestine beyond 200 cm. (6 feet 7 inches) is quite likely to result in

serious metabolic disturbances, which disappear, however, with the establishment of compensatory hypertrophy.

Experimental work on dogs has indicated that the rate of intesti-

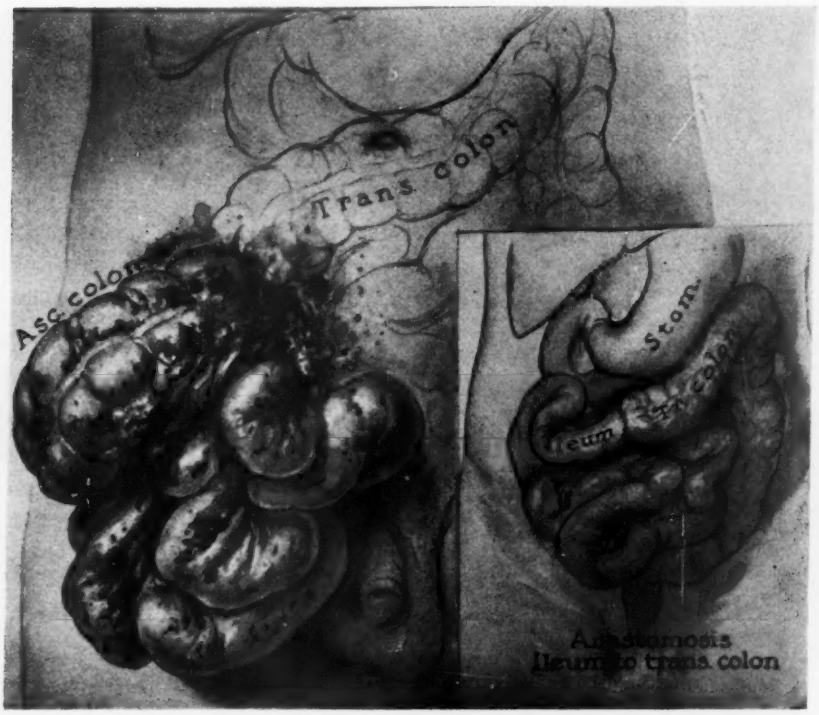

FIG. 1. Diagram of wound when patient was first seen. Insert: Postoperative appearance of anastomosis.

nal absorption above the site of resection is twice that of normal. The compensatory reaction, therefore, occurs entirely above the resection and there is no evidence to indicate that the colon or intestine distal to the resection aids in this substitution. In resection of the jejunum there is an increase in pancreatic secretion, but in general after such resections the remaining intestine works more efficiently in the absorption of carbohydrates and does its poorest work in the absorption of fats. Consequently, any patient who has been subjected to a massive intestinal resection will do best on a high carbohydrate diet and with a limited intake of fats.

REVIEW OF THE LITERATURE

The earliest recorded case of resection of a large amount of intestine is that of Koeberlé (1881) in which 205 cm. (6 feet 9 inches) of small intestine were removed from a woman, aged twenty-two,

for multiple strictures. Recovery was followed by no digestive disturbances.

In 1896, Ruggi reported a resection of 330 cm. (10 feet 10 inches)

FIG. 2. Postoperative *x*-ray, showing short loop of intestine remaining, also several shot still present.

in a boy, aged eight, who made a good recovery. The same year Fantino recorded the resection of 310 cm. (10 feet 2 inches) of ileum from a man, aged sixty, who had a large inguinal hernia with volvulus of the intestine and gangrene. He made a good recovery and had no complaints when seen one year later.

In 1900, Hayes resected 255 cm. (8 feet 5 inches) of ileum from a boy, aged ten, who recovered, but suffered from occasional diarrhea and vomiting and developed chorea. In 1901, Lauwer reported the resection of 265 cm. (8 feet 9 inches) of small intestine from a woman, aged sixty-five, for ventral hernia with intestinal obstruction from adhesions. She recovered.

In 1907, Edward Staehlin recorded a resection of 10 feet 2 inches of small intestine, and Littlefield, in 1919, resected 13 feet of the ileum with a good result. In 1923, Pirie Watson reported a resection of 7½ feet of the ileum three years previously, with the patient not only in good health but free from nutritional disturbances. In the

same year Jacob Sarnoff, of Brooklyn, reported a case in which he not only resected 15 feet of small intestine but also did a hysterectomy. The postoperative course was complicated by a partial

FIG. 3. Stomach fifteen minutes after barium meal; transverse colon is beginning to fill.

intestinal obstruction and fecal fistula but the patient eventually made a good recovery.

The longest resection on record is in a case of Von Brenner, in which 18 feet of small intestine was removed. Four other cases averaging 400 to 540 cm. (13 to 18 feet) are those of Gheden (534 cm.), Nigrisili (520 cm.), Axhausen (475 cm.) and Pouchet (400 cm.). In each instance the patient recovered from the operation but died shortly thereafter.

CASE REPORTS

CASE 1. This case was reported in detail by the authors before the Western Surgical Association in 1940.

Mrs. E. L., aged thirty-eight, para 3, was seen at the Graham Hospital, Canton, on the morning of July 6, 1939, with the complaint of severe abdominal pain. She had had many previous attacks of abdominal pain and

FIG. 4. One hour after barium meal. Colon is filled. Note exceedingly short length of jejunum between duodenum and site of anastomosis at transverse colon.

her appendix had been removed without relief of the symptoms. On examination there was a sausage-shaped mass, somewhat movable, extending from the right upper quadrant to the pelvis. A tentative diagnosis of paralytic ileus produced by a large retroperitoneal tumor was made. Because of an elevation of temperature the possibility of an infected right kidney was considered. The insertion of a Levine tube and Wangensteen suction gave no improvement, so operation was decided upon.

On opening the abdomen a large blue mass, too large to be delivered, appeared covered with peritoneum. The incision was enlarged, the mass delivered and found to be a thick, rubbery piece of intestine starting as an intussusception of the jejunum to 10 cm. (4 inches) from the ligament of

Treitz and involving a part of the ileum. The mass was about 15 cm. wide by 32.5 cm. long, and yet the mesentery was long enough, in spite of the edematous dense tissue, to permit the mass to be brought outside the

FIG. 5. Barium enema showing increased diameter of small intestine proximal to anastomosis.

abdominal cavity. Attempts to reduce the intussusception were futile, so the entire tumor was resected. Examination of the specimen showed that it consisted of nine layers of small intestine, that is, the intussusception was quadruple or "compounded" four times. It was 205 cm. (6 feet 10 inches) long. There were no adhesions present between the layers of the intussusception and no evidence of tumor, polyp or other cause could be found. There were large areas where the entire thickness of the wall was gangrenous, and the invaginated tissues were very edematous due to the long period of circulatory interference. The patient had a prolonged convalescence, leaving the hospital at the end of six weeks. She has been followed for three years. Roentgenogram of the stomach following barium meal revealed normal findings. She has no metabolic disturbance such as might be expected from the removal of so large a portion of the intestine.

CASE II. H. E., aged eighteen, entered the Graham Hospital on April 17, 1940, with a shotgun wound of the abdomen, accidentally inflicted while hunting. He was in severe shock and complained of abdominal

FIG. 6. Postoperative scar.

pain. In the right lower quadrant of the abdomen there was an opening 8 by 5 inches through which the intestine eviscerated. The eviscerated portion, which was bloody, dirty, perforated with shot and burned with powder, included the cecum, right half of the transverse colon, and many loops of ileum. A portion of the abdominal wall located over McBurney's point and above, measuring 6 by 4 inches, had been completely shot away.

The first impression was that the boy's condition was utterly hopeless. Nevertheless, sterile warm packs were placed on the eviscerated intestines, intravenous adrenal cortex was given, and intravenous glucose and saline started. He was typed for transfusion and found to be type 3. His father was type 4; however, 500 cc. of citrated blood was obtained from the father and added to the intravenous fluids. In about two and one-half hours he had recovered from the shock to such an extent that his temperature was 98°F., pulse 104, respirations 26, and blood pressure 95/65. His facial expression had changed from that of a moribund patient to one of mental alertness, and the transfusion had restored his color to some extent. It was thought that perhaps with a resection of a small amount of intestine the patient might have a chance for recovery.

Another transfusion of 500 cc. of citrated blood was started, and he was anesthetized with ethylene and ether. The gunshot wound was extended by an incision upward from the center of the wound, another one down along the right rectus muscle. Blood-stained fluid was present in the abdomen and considerable fecal matter had spread over the entire area. The damaged intestines were wrapped in sterile moist towels and the abdominal wall carefully cleansed with soap and water. No other skin preparation was carried out.

After redraping the wound, an examination was made to determine the extent of the intestinal damage. Since there were numerous shot lacerations of the bowel, both the intestine and its mesentery were damaged beyond any hope of repair, so the ileum was withdrawn from the abdominal cavity with the hope of finding an undamaged portion. He had been shot with a 12 gauge shotgun, the shell of which contained about 380 No. 6 shot, which will give some idea of the damage inflicted. Finally when the abdomen had been almost eviscerated, uninjured intestine was delivered which seemed to be the jejunum. Once more it appeared as though the condition must be hopeless, but as the patient's pulse was good and there was no place to stop, the small intestine was divided just proximal to the first shot perforation and all intestine distal to it was removed. Included in the resection were the remaining portion of the small intestine, the cecum which was badly lacerated, the ascending colon and hepatic flexure, which had only a few perforations, and the first half of the transverse colon, which had been practically torn in two. The end of the resection was just beyond the middle of the transverse colon. The mesenteric bleeding was controlled with hemostats and an end-to-end anastomosis was done between the small intestine and the transverse colon in two layers, the inner one of No. 00000 catgut and the outer layer of fine silk. The mesentery was sutured and the portion of the peritoneal cavity most extensively contaminated was irrigated with normal salt solution. The skin, fascia and muscle were carefully débrided until normal tissues were reached. The peritoneum could be closed satisfactorily, but the muscle and fascia had been so extensively removed that only the upper and lower angles could be sutured. An area of 5 by 4 inches of skin was left open and the defect filled in with a pack of sterile vaseline gauze, such as is used in the Orr treatment of osteomyelitis. The patient was returned to his room in surprisingly good condition, with a pulse of 126, respirations 24, and blood pressure 60/30.

The postoperative course was stormy and it is our belief that the patient was kept alive largely by transfusions twice daily. His highest temperature was 103.4°F. on the third postoperative day, and the highest pulse rate was 160 on the second day. The following morning he vomited, so a Levine tube was inserted and the Wangensteen suction apparatus applied. It was necessary to keep the tube in place for fourteen days, because each time it was removed the vomiting recurred. There was a considerable amount of serous discharge around the vaseline gauze pack; on the twelfth

day the pack was removed and replaced with a smaller one of the same type. He had no bowel movement until the third day when an enema was returned with a brown liquid and flatus. Following this, an enema every second day was returned with a brown liquid bowel movement. After the Wangensteen suction was removed, he was given a liquid diet of fruit juice and carbohydrates, and began to gain in weight and strength. As soon as his food intake was increased he developed a diarrhea which was checked by restricting the diet to fruit juice and carbohydrates. He was dismissed from the hospital the tenth week, weighing ninety-seven pounds, a loss of forty pounds from his normal weight. The wound was in good condition, with an area, 2½ by 3 inches, still uncovered.

The resected intestine measured 14 feet 8 inches with 22 inches of colon, a total of 440 cm.

Eight days after leaving the hospital he returned complaining of recurrent abdominal pain associated with diarrhea and a loss of five pounds in weight. On inquiry it was found that he had returned to his work as a fisherman and had partaken of fried fish on several occasions. Small amounts of tincture of belladonna were given and he was returned to a diet of fruit juice and carbohydrates, with no fats excepting small amounts of butter and cream with other food. Within two days the diarrhea was controlled; the stools became somewhat more solid but were still very largely liquid. He was then given six feedings a day of rather limited quantity and soon began to gain weight. At the end of one month he was discharged from the hospital, with the wound healed, and an average of two soft or semiliquid bowel movements daily. He was much improved in strength and his weight was 103 pounds.

He returned for a check-up three months later, in November 1940, weighing 117 pounds. His diet was restricted largely to carbohydrates with a small amount of finely chopped meat and very limited intake of fats. Vitamins were furnished artificially and with the fruit juice. The bowels moved three times a day and the movement was usually liquid but occasionally semisolid. He was gaining in strength and considered himself in good health.

Nonprotein nitrogen was 32 mg. per 100 cc. of blood, and the fasting blood sugar was 104 Gm. per 100 cc. The blood count was within normal limits as was the urinalysis.

Roentgenographic and fluoroscopic examinations were made of the gastrointestinal tract. It was interesting to note that the barium meal upon leaving the stomach appeared to go through one short loop of intestine and enter immediately into the transverse colon. Not more than two or three feet of small intestine remained, but in spite of this loss his condition seemed remarkably good.

The patient was last seen on May 5, 1942. Roentgenographic examination showed the stomach quite enlarged and it emptied in three and one-half hours. The jejunum was also larger in diameter than is normal and

emptied directly into the transverse colon. The jejunum had enlarged and apparently had taken over the function of the ileum, and under the fluoroscope appeared as only a single loop of bowel. The barium meal on leaving the duodenum passed through this loop and into the colon, and the loop did not appear to be more than three feet long.

The abdominal wall was quite solid. The scar was thick and had a keloid appearance, such as is commonly seen after an extensive burn. He was in an excellent state of nutrition and was able to do the heavy work of a fisherman. He stated that abdominal cramps followed the eating of fried or greasy foods but no diarrhea occurred.

Blood chemistry examination showed blood sugar 105 mg. per 100 cc. of blood and nonprotein nitrogen 22 mg. per 100 cc. of blood. Urinalysis and blood count were within normal limits.

SUMMARY

Experience with these two cases brings out the following facts:

1. Massive intestinal resection can be performed with favorable results even under extraordinary conditions, such as were present in the second case.

2. The pre- and postoperative care played an important part in the final outcome of both cases.

3. The second case verified statements made in the literature that the diet after such resection should be high in carbohydrates and proteins, but low in fat and cellulose.

4. The author's experience with two cases of massive resection with recovery indicates that even in what appears to be a hopeless situation it is worthwhile to make every effort to save life.

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BRACHIAL PLEXUS BLOCK ANESTHESIA OF THE UPPER EXTREMITIES*

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THERE have been few recent reports in the American literature on the use of brachial plexus block anesthesia. The impression obtained from conversations with many leading surgeons is that the percentage of unsatisfactory anesthesia is so high in most hands that this procedure is used only by a few who have perfected the technic. The procedure most generally used has been the supraclavicular route as described by Kulenkampff, quoted by Labat.[1] Previous personal experience with this older technic was unsatisfactory and the procedure was abandoned until Patrick[2] described a modification which has been eminently successful in our hands. The method of Kulenkampff and Labat depended upon the disposition of the solution at exact anatomical points on the plexus. These points were difficult for the novice or even the expert to reach with the tip of the needle, particularly in obese patients. Patrick's modification depends upon laying a wall of anesthetic solution from the first rib out to the deep fascia at right angles to the path of the plexus as it passes about the rib. Exact localization is unnecessary, as the solution diffuses through the fascial compartment occupied by the plexus. This technic has in our hands reduced primarily unsuccessful anesthesia to 3 per cent.

Premedication is of primary importance not only to allay the patient's apprehension but to protect against procaine toxicity. A barbiturate, usually sodium phenobarbital (.130 Gm. for 150 pounds of body weight) is given hypodermically thirty minutes before the block is started, or if time is pressing, one-half of the above dose is given intravenously. The patient is placed on the table, supine, with a thin pillow beneath the upper dorsal spine, the shoulder on the involved side is depressed, the arms placed at the side and the head turned toward the opposite side.

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The surgeon determines by palpation, the midpoint of the clavicle, between the sternoclavicular and the acromioclavicular joints. An intradermal wheal of novocaine is raised ½ cm. superior to the midpoint of the clavicle. A No. 22 gauge needle three inches in length and not attached to the syringe is then inserted, inward, downward and backward, and roughly toward the second or third dorsal spine, until the first rib is encountered well posterior. If blood is encountered at any time, the needle is withdrawn and reinserted at a slightly different angle. The needle after encountering the first rib is withdrawn 2 to 3 mm. and with a glass syringe 10 cc. of 2 per cent novocaine is injected as the needle is withdrawn to the level of the deep fascia. It is then reinserted at a slightly different angle with the needle point more lateral and anterior but still contacting the first rib, and 10 cc. of solution is injected as the needle is withdrawn. The object in withdrawing the needle point is to enable permeation of the nerve trunks of the plexus. It must be recalled that the brachial plexus and the subclavian artery lie like meat in a sandwich between two fascial planes: in front, a layer of fascia extending across from

TABLE 1

		Simple	Compound
I. Reduction of Fractures:			
Colles's		2	0
Ulna and radius	U/3	4	0
	M/3	11	2
	L/3	4	1
Radius	Head	1	0
	U/3	1	0
	L/3	5	0
	Resection of head	3	0
Humerus	U/3	0	1
	M/3	1	1
	L/3	1	3
Metacarpals		1	2
Phalanges (multiple)		1	1
Navicular		1	0
Traffic elbow		0	1
Open reduction	Olercranon	4	0
	Metacarpophalangeal, joint	1	0
		41	12
II. Primary Tenorrhaphy:			
Biceps L/3			1
Triceps L/3			2
Forearm and wrist			7
Fingers			8
Hand			4
			22

TABLE 1—(*Continued*)

	Compound
III. Débridement, Irrigation and Suture of Lacerated Wounds:	
Elbow	4
Forearm	5
Hand	1
	10
IV. Débridement, Irrigation and Suture of Gunshot Wounds:	
Forearm	2
Hand	1
Humerus M/3	2
Fingers	1
	6
V. Amputations:	
Arm M/3	1
Forearm M/3	1
	2
VI. Miscellaneous:	
Ganglionectomy of wrist	1
Incision and drainage of abscess of web space (severe diabetic)	1
Excision palmar fascia (Dupuytren's)	1
Hemisection of hand	1
Removal of screws from graft on radius	1
Débridement human bite of hand	1
Excision tuberculoma of elbow	1
	7

the scalenus anticus, and behind by a layer over the scalenus medius. By withdrawing the needle during the injection this compartment is traversed and a triangular wall of solution is deposited, based on the first rib with the apex of the triangle at the wheal.

The needle is reinserted some four to six times, always through the initial intradermal wheal advancing the needle point laterally and finally medially along the first rib from back to front. No attempt is made to encounter or provoke paresthesias. However, the patient is told to expect pain running down the arm especially to the little finger and to tell the surgeon. If such a pain is encountered, a single injection is made without moving the needle, directly into the plexus. There are anatomically no dangers laterally, but as the medial and anterior position is approached the subclavian artery must be missed by the needle point. The lower trunk of the plexus lies deep to the artery and must be injected to assure a perfect block. When the needle is inserted at its most sternal portion, it is seen to pulsate with transmitted beats from the subclavian artery. It is here that the final injection is made. Often the presence of the artery is

first made evident by blood exuding from the needle. If this happens, remove the needle and reinsert at a slightly different angle. At no time despite several entrances into the vessel has there been any hemorrhage from the vessel or any other untoward complication. The amount of 2 per cent novocaine was arbitrarily set at 60 cc., as the maximum dose for a 150 pound man without chancing immediate or delayed novocaine reactions.

The block is given with the surgeon wearing sterile gloves. Fifteen minutes should elapse before starting the operation after completing the injection. This period is used for the surgeon to scrub, don gown and gloves and prepare the field of operation. By this time anesthesia usually is complete.

The blocks reported in this series are 100 consecutive cases done in the Louisville City Hospital (University of Louisville School of Medicine) by the surgical resident staff from July, 1940, to October, 1941. A total of eighteen house officers contributed their cases. Several of them performed only one case and the largest number of cases by one man (W. H. W.) was twenty-nine. It is interesting that eighteen cases in this series represent the surgeon's initial case.

The cases selected for brachial blocks were those of trauma to the upper extremity. There were fifty-three reductions of fractures of which forty-one were simple and twelve compound. Of the simple fractures, eight had open reduction. The fractures ranged from multiple phalangeal fractures to a compound fracture of the upper third of the humerus. Twenty-two primary tenorrhaphies were done in the lower third of the arm, forearm, wrist, hand and fingers. Ten lacerated wounds of the elbow, forearm and hand were débrided, irrigated and sutured. There were six gunshot wounds of the arm, forearm, hand and fingers that were débrided, irrigated and sutured. Two amputations, one through the middle third of the arm and the other, the middle third of the forearm, were done. A miscellaneous group of seven cases consisted of: (1) ganglionectomy at the wrist; (2) incision and drainage of an abscess of the web space of the hand in a severe diabetic; (3) excision of palmar fascia for Dupuytren's contracture; (4) hemisection of hand; (5) removal of screws from graft on radius; (6) débridement of human bite of hand; (7) excision of a tuberculoma of elbow.

No attempt was made to measure the length of time that the anesthesia lasted, but the average length of 100 operations done under brachial block alone was sixty-seven minutes. The shortest operation required three minutes. This was an incision and drainage

of a web space abscess of the hand in a severe diabetic. The longest case required 195 minutes and was a tenorrhaphy on the finger flexors. As would be expected in a traumatic series such as this the males predominated the females 77 to 23. There were fifty-nine white cases as compared to forty-one negroes. The average age of the patients was thirty-two years. The youngest patient was an eight-year old white girl who had a reduction and cast for a fracture of the middle third of the ulna and radius, the procedure lasting sixty minutes. A total of five nine-year olds had forearm reductions or tenorrhaphies. The upper age limit was represented by two men aged sixty-seven, one of whom had a compound traffic elbow and the other a severe laceration of the forearm and cubital fossa. The former operation lasted 140 minutes and the latter 150 minutes.

Of the 100 cases three were initial failures. It is interesting to note that one of these patients had an excellent motor paralysis but had a poor sensory loss. One of these represented the first block attempted by a member of the house staff. The third failure also developed a small pneumothorax on the same side due to poor technic. A second case developed a 25 per cent pneumothorax with excellent anesthesia of the upper extremity. Neither of these complications were of any significance. Two patients had mild procaine reactions that were easily controlled with intravenous sodium phenobarbital. One of these had received no preoperative barbiturate. In one case cyclopropane was used after fifteen minutes of successful block because of the unco-operativeness of the patient due to alcoholism. Only eight cases required some type of supplementary anesthesia because of the wearing off of the block anesthesia before completion of the operation. The shortest time a block lasted was sixteen minutes and the longest was eighty minutes before supplementary anesthesia was required. All cases have been followed in postoperative and fracture clinics and there has been no evidence of any delayed complications.

SUMMARY

1. A technic of supraclavicular branchial plexus block anesthesia is described and a consecutive series of 100 cases reported with three failures.

2. The operations performed under this type of anesthesia are presented.

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THE TRANSVERSE PALMAR INCISION

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THE transverse palmar incision is not new. It has been in use in the Montreal General Hospital for at least thirty years. Its originator is not known. It was described by the author in 1926[1] and was published by him in 1928.[2]

The many advantages of this incision, and its practically total lack of disadvantages, are, I believe, sufficient excuse for bringing it to the attention of the profession.

Some of its more important advantages are: (1) Complete and adequate exposure in infections of the palm of the hand ("collar button," web or lumbrical space infections and midpalmar space abscess); (2) ease of combining with the lateral finger incision for suppurative tenosynovitis; (3) ease of enlargement to secure complete relief of tension, if necessary across the whole width of the palm; (4) complete freedom from danger of postoperative, or post-infective finger flexion deformity, similar to the Dupuytren's type; and (5) an almost invisible and totally painless scar.

Like all others in the treatment of hand infections, this incision should be made in a "bloodless field." This implies the use of general anesthesia. Insistence on these two elemental points should be unnecessary.

TECHNIC

The incision should not be made too far proximally. About one-quarter of an inch from the proximal transverse crease at the base of the finger is usually the correct site. The two fingers near whose bases the maximum lesion is present are slightly separated and a small incision is deepened until pus is encountered. A director is then passed transversely until the limits of the cavity are reached. This will be found to be superficial to all important structures such as tendons, blood vessels and nerves. The cavity may then be explored with the tip of the finger and all necrotic material wiped out with gauze. (Fig. 1.)

The frequent occurrence of palmar infections in conjunction with, or as a complication of, suppurative tenosynovitis of the flexor

sheaths of the fingers, makes necessary the description of the lateral finger incision. The ease of combining the two incisions has already been referred to.

FIG. 1.

FIG. 2.

The principles on which the *lateral* finger incision is based are, curiously enough, almost identical with those in favor of the *transverse* palmar incision. The main one is the avoidance of placing a scar at right angles to a flexion crease.

The finger is flexed and the incision is made at a point just dorsal to the termination of the interphalangeal flexion crease. It should be begun on the lateral aspect of the proximal phalanx and continued distally and proximally as is necessary. Care should be taken not to damage the capsule of the interphalangeal joints. The incision should be carried out with the utmost care in the proximal direction and should not be allowed to approach any nearer to the palmar surface of the finger, but should be carried along the same plane to the web. When this point is reached, it is connected with the palmar incision by cutting through the web at a point exactly midway between the fingers and at right angles to the palmar incision. Failure to observe this latter point is about the only way damage can be done to the digital vessels and nerves. This is well shown in Figure 2 in which the combined incisions are seen with the vessels and nerves safely entering the anterior finger flap. The possibility of their damage by a careless "corner cutting" procedure should be quite evident.

The author will, he trusts, be pardoned for a word in defense of the much condemned "fish-mouth" incision which happens to be shown in the illustration. Practically all the unfavorable results ascribed to its use are due to its being made too far anteriorally. If it is made quite close to the nail, the question of a scar on a pressure-bearing surface will not arise.

Use of the B. I. P. P. and liquid paraffin and curtain drainage method as described in the original article[2] and in later publications[3,4] has, over a period of years, served to convince the Author of its superiority to all others, in the treatment of hand infections, and most particularly in connection with the incision just described.

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IMPACTION OF FRACTURES BY LARGE PRESSURE TONGS

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THERE are some types of fractures that can best be treated by forcing and molding the fragments back into position. The most common are the fractures of the os calcis and the proximal end of the tibia. Other fractures may be treated in this manner but they are not common.

FIG. 1. Large pressure tongs three feet long; hinge seven inches from blade end; blades padded with felt.

The os calcis is usually fractured by a fall from a height which produces a displacement upward with a broadening of the body of the bone so that it impinges on one or both malleoli. If this broadening is not corrected, there is a persistent deformity which causes pain on walking. By reversing the mechanism that produced the fracture the fragments can often be molded and forced into their normal position and will remain so if the foot is placed in a cast.

A quick and easy way to accomplish this maneuver is by the use of large pressure tongs with padded blades as shown in the illustration. A firm squeeze on the os calcis just below the malleoli combined with traction will often give a very satisfactory reduction. The tongs are made large with long handles and padded blades so that considerable quick pressure can be used without much effort, and no

FIG. 2. FIG. 3. FIG. 4.

FIG. 2. Apply firm pressure below malleoli to squeeze bone into position using counter-traction behind heel and against sole.

FIG. 3. Tongs can be applied to outside of cast to make gentle pressure while cast is hardening.

FIG. 4. Tongs are used over the head of the tibia to push the fragments together.

FIG. 5. Fracture of os calcis; before and after use of pressure tongs.

FIG. 6. Fracture of tibia; before and after use of pressure tongs.

damage is done to the soft parts. A small pad of felt may be placed just below the malleoli after reduction and incorporated under the cast. Slight pressure may be applied to the cast over the felt pads with the tongs while the plaster sets so as to insure a snug fit over the sides of the os calcis below the malleoli.

The manipulation is best carried out under an anesthesia, such as morphine-scopolamine, as soon as possible after the injury and before much swelling has occurred.

The cast should extend from the toes to below the knee. In more extensive fractures it may be necessary to extend the cast onto the thigh with the knee flexed to a right angle so as to keep the tendon Achilles relaxed.

Some fractures of the proximal end of the tibia and other bones are of such a type that a firm quick squeeze will force the fragments into their normal position and they will remain so without any other treatment than fixation.

The first large pressure tongs we used were hammered out by a blacksmith in 1928 and have been used frequently. We present the idea now as another method that may be used to advantage in the treatment of a selected group of fractures.

A PLEA FOR CAREFUL EXAMINATION AND EARLY EVALUATION OF TRAUMATIC CASES

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THE traumatic surgeon should have an abundance of time to give to each injured patient. Every traumatic case should be carefully examined under proper surroundings as soon as the patient's condition will permit. Even the slightest trauma should receive careful consideration, since minor injuries may not only produce disability and deformity but also end far too often in death. An injured person is entitled to a most careful and complete examination with all pertinent findings being recorded. The best obtainable consultants should be brought into the case early, when group consultation is desirable. This will be fairly frequent, as many traumatic patients are too seriously injured for a lone surgeon to contend with. A traumatic patient carefully examined with the findings recorded is more certain of proper treatment, and consequently has better chances for a satisfactory recovery.

In no other field in medicine does so much depend upon repeated *x*-ray examination for diagnosis and correct evaluation. Skilled and discriminating *x*-ray interpretation, based upon faultless technic, can do much for a better solution of the many problems involved in the evaluation of disability. Roentgen examination should follow the clinical investigation, unless bone injury can be absolutely excluded by the report of the accident and the clinical findings; otherwise, regardless of the extent and nature of soft tissue damage, *x*-rays should be taken early soon after the accident. As fractures frequently are unrecognized, an early evaluation may be incorrect. No guessing is to be tolerated in any case.

The difficulty in evaluating lower back disability, that greatest bugbear of industrial practice, arises out of one, or it may be all, of four failures on the part of the practitioner: failure to know minutely the anatomical structure of the lower back and the anomalous variations so frequently found; failure to obtain a detailed and correct history of the mechanism of the injury; failure to have an adequate

x-ray examination; failure to guide carefully and gradually the rehabilitation and re-education after a period of immobilization.

All of these four points are essential. In particular the consulting surgeon on traumatic cases is often astounded at the rashness with which both positive and negative conclusions are drawn from inadequate *x*-ray examinations. The majority of cases have only anteroposterior and lateral views, which are not sufficient; many instances are seen in which only anteroposterior flat films are made and conclusions are drawn from these alone. Not only are oblique views necessary, in addition to anteroposterior and lateral views, but many injuries to the lumbosacral area should have films taken in upright as well as supine posture. In addition the lumbosacral area requires films taken at a different angle from the remainder of the lumbar spine; consequently, the usual films of the lumbar spine as a whole are not adequate for the lumbosacral region. Stereoroentgenograms are frequently indicated. Finally, study and interpretation by a trained and qualified roentgenologist is most desirable, unless the industrial surgeon is so qualified.

In every disabled traumatic case there are factors which are immeasurable, imponderable and intangible. The traumatic surgeon owes it to his patient to see that an accurate estimate of his disability is made early. Not only has the traumatic surgeon a serious duty in estimating the disability sustained by a patient, but also a definite responsibility toward the insurance carrier; therefore, he must be strictly impartial. The end results deserve first consideration. A diagnostic or therapeutic error may be responsible for prolonged treatment and for a high degree of permanent disability with all its implications for the injured person as well as for the insurance carriers.

The traumatic case is not a machine to be repaired but a human being to be helped. No two persons are alike in thought or action, in temperament, personality or emotional reactions. Therefore, the examiners must listen to the patient attentively, observing him carefully to obtain a correct estimate of his attitude toward recovery and his desire or willingness to return to work.

Though several prominent traumatic surgeons—Keisler, McBride, and others—present very practical methods for evaluation of traumatic cases, there is no substitute for training, experience and sound judgment.

Whatever method may be used to evaluate a disability, it is usually found that the simpler the method, the better the results.

The necessity for some guide, however, in determining the average period of incapacity for given clinical conditions is apparent. Studies of nonlitigation cases furnish a useful basis for establishing average periods of incapacity, but these averages must be considered as the irreducible minimum.

The attitude of the physician treating the traumatic patient will materially influence the mental attitude of the patient. Patients told by their attending surgeon that their injuries are unusually serious will have an unfavorable outlook toward recovery, as compared with the patient who has been encouraged and told that he will make the best recovery possible. Recovery may be retarded when the attending surgeon overemphasizes caution. Patients in a ward with dissatisfied traumatic patients should be moved to new quarters whenever this condition is discovered. Again, patients in contact with dissatisfied friends and relatives will be influenced unfavorably toward recovery. Everyone about the patient should be asked to help build up a healthy attitude toward recovery in order that an early evaluation can be made. Also forewarn against discouragement upon resumption of work and encourage perseverance. The compensation patient, paid for his disability, is likely to prolong his complaint of disabling symptoms, whereas the noncompensation patient will make an effort to return to work as soon as possible. He will often put up with the inconveniences occasioned by an injury, whereas the compensation patient will expect to be rid entirely of all inconvenience before he returns to work.

An accurate and detailed history is needed not only of the accident in question but of any diseases or former accidents; only in this way will a definite visual reproduction of what actually took place be reproduced in the mind of anyone hearing or reading the description.

Regardless of whether the case is compensable, the traumatic surgeon and his consultants should evaluate the injuries and their results as early as possible. They should determine the direct and proximate result of the injury, and the length of time necessary to relieve the condition. They should estimate the probable outcome; how long it will take the injury to reach its final stage of healing; whether the disability will be temporary, with complete restoration of function; or permanent; and finally, what per cent of the permanent loss of function has resulted from the injury. Experience proves that many traumatic cases have definite disability not related to the injury; nevertheless, symptoms resulting from such disability often are attributed to the accident by the patient. Frequently, he may

believe such is really true; therefore, the physician must determine how much of the disability developing subsequent to an accident is actually due to that accident. This determination can be made only when competent medical observation has decided how much of the disability is the natural result of the disease and how much added disability has been produced by the accident. The rational approach would seem to be to attempt to decide what the natural progress of the disease would have been without the accident and then to add the effect of the accident to this. A systematic, careful, detailed and intelligent study of each individual case is the only procedure which will bring satisfactory results. Each case is a problem unto itself and should be studied from every aspect, historical, anatomical, physiological, pathological and psychological, utilizing every resource of clinical and laboratory analysis.

The traumatic surgeon and consultants, therefore, when evaluating traumatic cases, should obtain a history of the accident, the progress of the case up to the time of evaluation, and the symptoms complained of at the time of the examination; all of this should be recorded. Then they should make a detailed and painstaking physical examination, carefully recording the complete findings. Finally, they should examine such laboratory records, including roentgenograms, as are at hand and order such other examinations as are deemed advisable to secure the necessary data.

Many problems must be considered in the evaluation of traumatic cases. Unfortunately, the period of temporary disability as well as the percentage of partial permanent is influenced by the factors of compensation and noncompensation. The mental attitude of the injured person toward his disability and his inclination to return to work are strong factors in each individual case. The traumatic patient's concept of himself and the outer world frequently undergoes a drastic change unless an effort is made to bolster his morale. The traumatic surgeon will find that the proportion of patients presenting mental symptoms of etiological significance during their illness is much larger than is generally believed. Since, as is well known, purely emotional causes, if severe and prolonged, may produce structural changes, the physician should guard against a traumatic neurosis in his patients, using every means to prevent it. The onset of symptoms indicating this condition means there is reason for haste in evaluating the disability. The treatment of traumatic neurosis, so-called, in the acute stage is prophylactic.

Therefore, the earlier the evaluation, the quicker and more certain will rapid recovery occur.

One must remember that anatomic repair does not mean functional restoration is fully established. Sufficient time must elapse to allow the ordinary consequences of trauma to disappear by natural body means. The injured person need not wait, however, for the maximum adaptation, but through re-education to altered conditions, return to work and endeavor to obtain maximum efficiency. One with a physical handicap must, therefore, develop new habits of use so that improvement through adaptation is not final until the injured part can be used without conscious effort. The attending surgeon should foresee such conditions and explain to the patient what is to be expected, thereby preventing discouragement and dissatisfaction with the evaluation of the disability.

CONCLUSIONS

1. After taking part in more than 1,200 industrial consultations, it is my opinion that traumatic patients require a very careful examination for a correct diagnosis, to get the best treatment possible, to reduce both temporary and permanent disability and to obtain an early evaluation.

2. A consultant or group consultation is advisable for correct diagnosis and exact evaluation. Such service should be assigned to the most thoroughly trained and experienced traumatic surgeon and specialist in other fields of medicine having a knowledge of traumatic cases.

3. Properly executed roentgen examination should follow the clinical investigation. Skilled and discriminating *x*-ray interpretation by a thoroughly trained and experienced roentgenologist is most desirable as an aid to diagnosis and correct evaluation.

4. The mental attitude of the traumatic patient toward his disability and inclination to return to work is materially influenced by a number of factors, especially by the attitude of the attending surgeon. Giving expectation of improvement will result in a more rapid recovery and an earlier evaluation.

5. An early determination of the degree and type of the trauma is a prerequisite to the early and correct evaluation of disability.

6. Early evaluation insures the elimination of uncertainty on the part of the patient, a more speedy recovery, and an earlier return to work.

ADJUSTABLE TRACTION SPLINT FOR UPPER OR LOWER EXTREMITY

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Diplomate of the American Board of Surgery; Chief of Staff, Nix Hospital; Attending Surgeon, Santa Rosa Hospital

DUE to limitations and priorities on metal necessarily used in making Thomas or Murray-Jones splints, etc., I have devised this adjustable wooden traction splint applicable for splinting

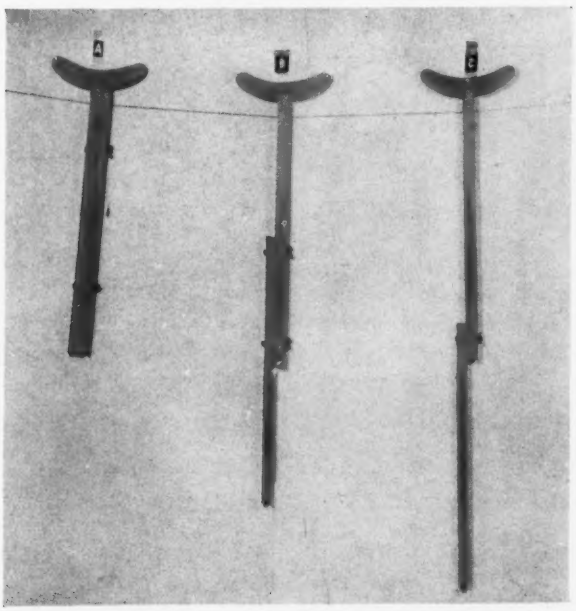

FIG. 1. A, splint closed; B, splint extended for traction, upper extremity; C, splint extended for traction, lower extremity. Because of tongue-groove principle, splint needs only one bolt.

either extremity in traction. The one piece fixed to the "crutch" is grooved while the movable piece is tongued so that it may slide (tongue and groove principle), from an over all of 25 inches to be locked by one or two bolts at any desired length up to 45 inches, when it is perfectly rigid against rotation as well as bending.

In applying traction to a fracture in the upper extremity the "crutch" is placed at the junction of the pectoral muscle and chest wall with a hitch about the base of the hand which is fixed over the

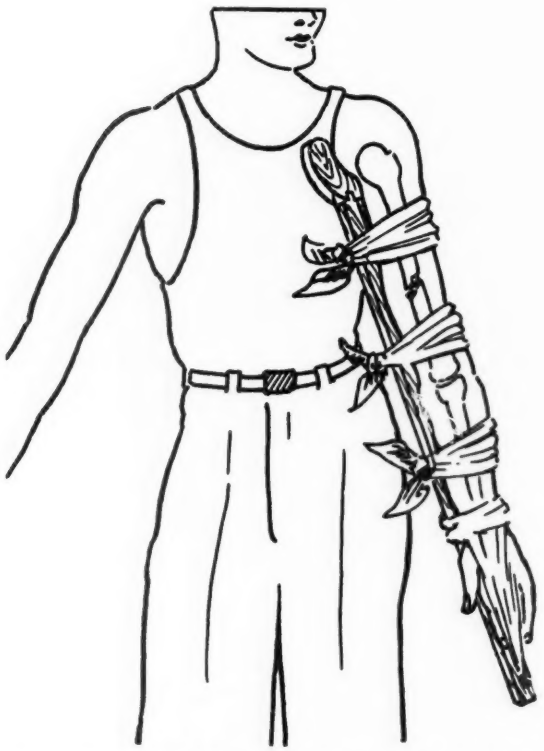

FIG. 2. Splint applied with traction and fixation in fracture of humerus. Note slight abduction with pressure of crutch against pectoral muscle. The tighter the traction, the greater the abduction.

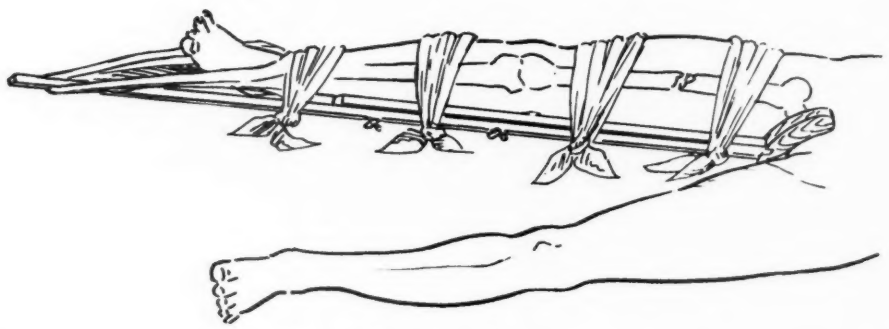

FIG. 3. Traction and fixation in fracture of lower extremity. Fragments are well mobilized for transportation.

distal end, which is notched, so that the greater the traction the more the abduction of the extremity. This position prevents "crutch" paralysis. A bandage, if available, or one or two handkerchiefs, etc., will stabilize the arm to the splint.

In fractures of the lower extremity the "crutch" is placed in the crotch, when with a hitch about the ankle sufficient traction is easily had. The limb is then stabilized to the splint with two or three ties of some sort.

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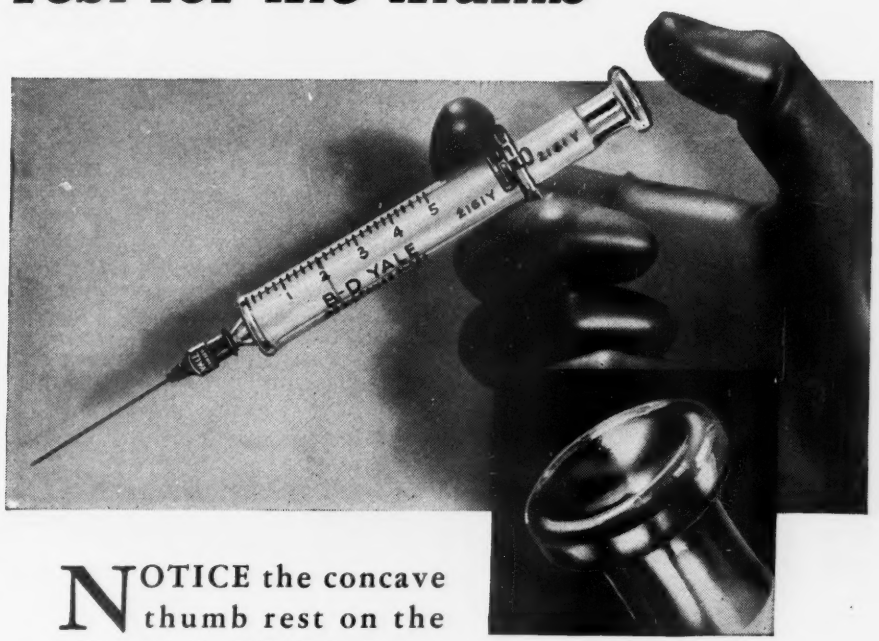

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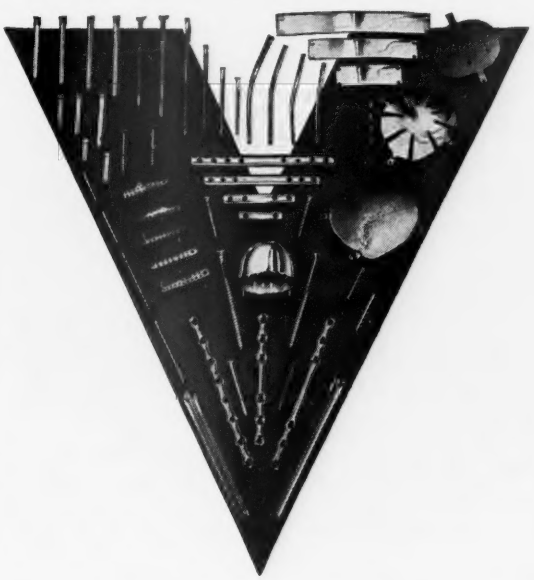

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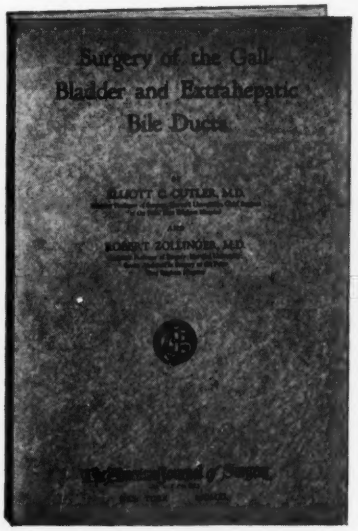

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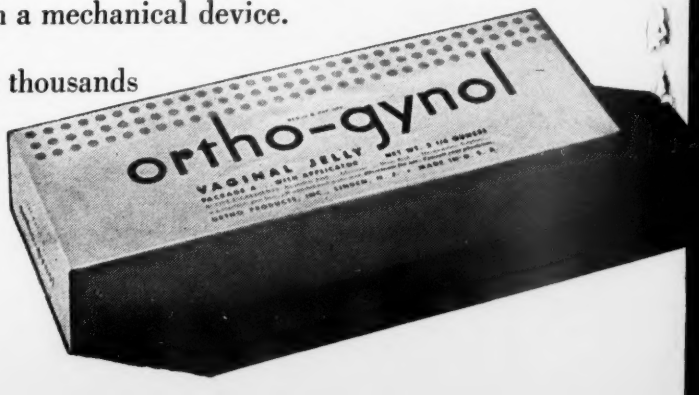

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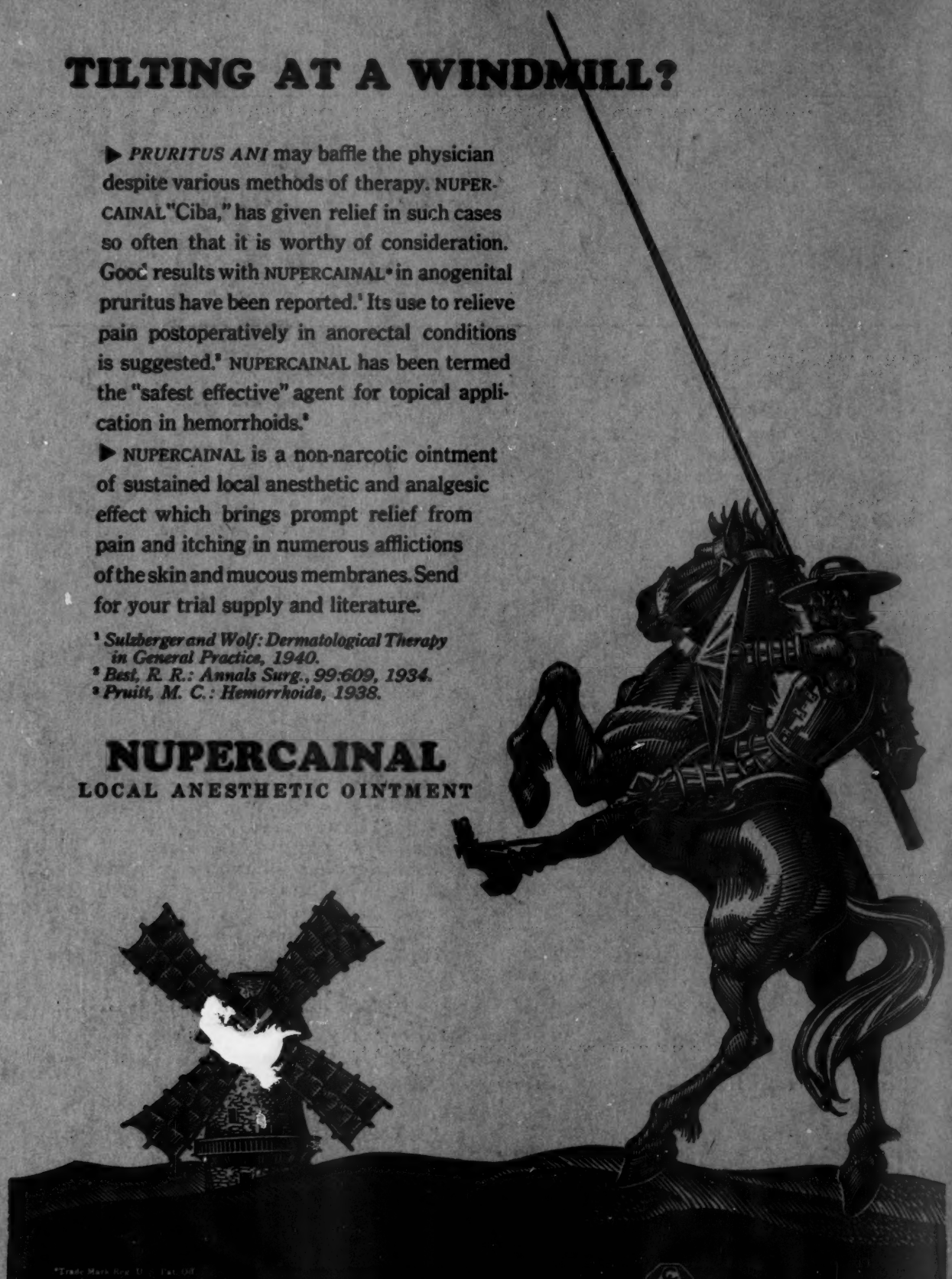

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